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THE UPPER ESOPHAGEAL SPHINCTER AFTER LARYNGECTOMY.*†

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The esophagus is shut off from the pharynx by an air-tight sphincter known as the cricopharyngeus. Although this sphincter was first described by Valsalva[1] in 1717, it remained for the anatomists and physiologists[2-6] of a later era to delineate and describe the functions of this structure.

Ingelfinger[7] quite adequately reviewed the literature pertaining to this sphincter in 1958.

The role of the parasympathetic motor supply to the cricopharyngeus, in the dog, was demonstrated by Kirchner[8] in 1958. The role of its sympathetic supply has not yet been determined.

There is little doubt that the cricopharyngeus acts as a true sphincter.[9,10]

The cricopharyngeus muscle, as visualized by the anatomist and the radiologist, is a structure measuring 1 to 2 cm. in length. Batson,[11] in 1955, in an unusual specimen in which this structure was remarkably well developed, reported the

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length as 1.2 cm. Measurements of intraluminal pressures, however, reveal an area of increased pressure of over 4 cm. in length. It is felt that both cricopharyngeal and esophageal muscles contribute to the sphincter mechanism.[12]

The function of the cricopharyngeus sphincter in the normal individual is usually thought to be that of a barrier to the free passage of air to and from the esophagus with respiration. In the laryngectomized patient, however, it is assigned a more significant role. It is thought by many to serve as the vibrating mechanism that produces sound, and it is believed that the more normal the sphincter, the better the speech.[13-17] The emphasis has been to reconstruct a tight sphincter in the laryngectomized patient at the time of surgery. The most difficult feature in mastering esophageal speech, however, is not in forming sound, but rather in acquiring the ability to swallow and regurgitate air. It appeared to us that a tight sphincter would make this procedure more difficult.

The present work was undertaken to determine whether a correlation existed between the ability of a laryngectomized patient to speak and the function of his cricopharyngeal sphincter.

METHODS AND RESULTS.

Measurements of esophageal pressures were made upon 15 normal and 20 laryngectomized patients. Of the laryngectomized patients, two were females and 18 were males. Their ages varied from 41 to 80. The interval between operation and the pressure studies varied from 28 days to slightly more than five years. Four patients had good and two had excellent esophageal voices. In six, the interval since operation was too short to determine whether they would be able to master an esophageal voice.

In performing the studies, the patients swallowed a tube made of three polyethylene catheters that were fused together and sealed at their tip (see Fig. 1). A lateral hole larger than the diameter of the catheter was made in each catheter at a distance of 0, –5 and –10 cm. from a reference point.

The entire tube was graduated in centimeters from the zero point or first lateral orifice.

The tube was swallowed by the patient to any desired depth. Measurements were made in centimeters from the incisor teeth to the lateral orifice. The tubes were then filled with water so that any pressure change within the

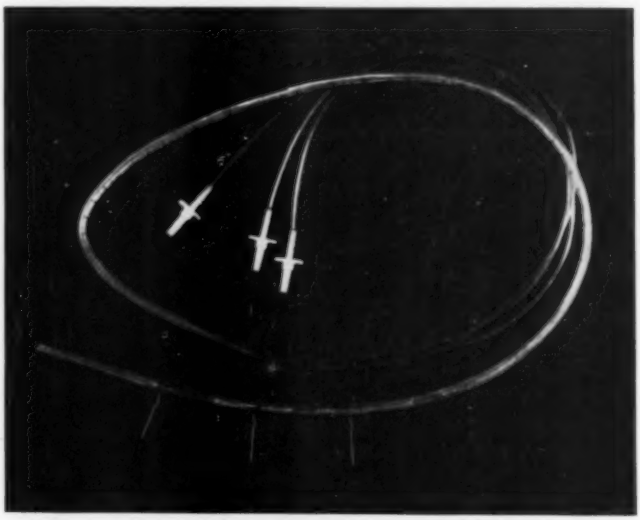

Fig. 1. Triple lumen tube used in measuring intra-esophageal pressures. Arrows mark the side openings, 5 cms. apart.

esophagus could be transmitted to transducers and eventually to a Sanborn direct writing recorder (see Fig. 2). The act of swallowing was signaled on a fourth channel by a transducer connected with the accessory muscles of deglutition.

The efficacy of this system has been demonstrated by the work of Ingelfinger,[7] Code,[10] Nagler,[16] etc.

RESULTS.

Pressure studies were made upon 15 normal subjects and

upon two cancer patients prior to laryngectomy. A typical normal esophogram is shown in Fig. 3. In this, and the succeeding figures, the uppermost line, A, is the myogram, in which the spike signals the act of swallowing. 1, 2 and 3 are recordings of the pressures at the orifices in the three catheters. 1 is the uppermost orifice, and 2 and 3 are 5 and 10 cm. respectively below 1.

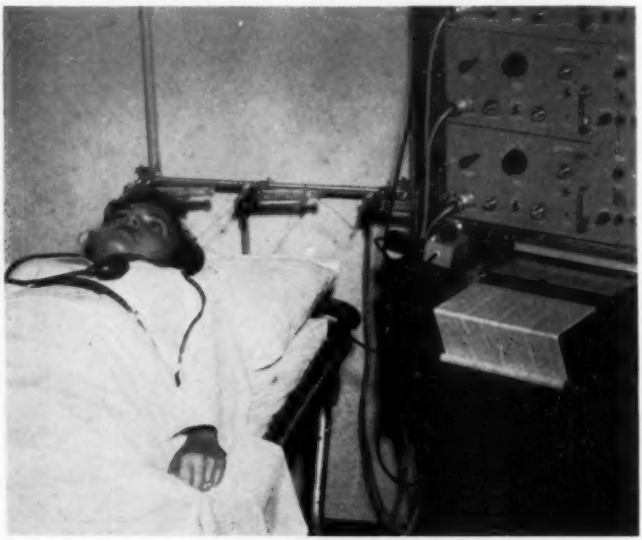

Fig. 2. Patient with tubes in esophagus. Transducers are above her head. Sanborn recorder at right.

The paper is calibrated in millimeters. The paper speed is 2.5 cm. per second. The machine is calibrated so that a deflection of one millimeter on the vertical is equal to one millimeter of mercury. The base line, or atmospheric pressure, is adjusted to coincide with the 15 millimeter bar before the start of the experiment so that negative deflections might be recorded upon the calibrated portion of the paper.

The strips presented in each figure are cut from significant portions of the tracing.

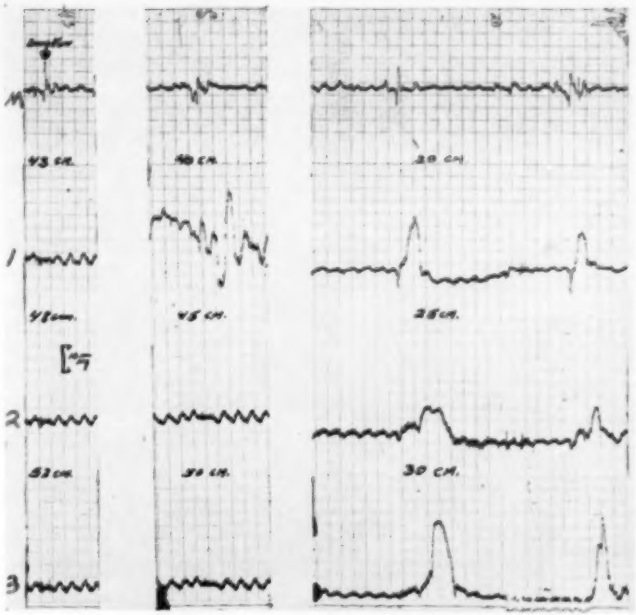

Fig. 3. Normal pressure recordings from various levels.
M—Myogram, signalling swallow.
A—All catheters in stomach.
B—Catheter #1 in lower esophageal sphincter. Nos. 2 and 3 in stomach.
C—All catheters in esophagus, #1 just below cricopharyngeus. Peristaltic wave seen at all 3 levels.

In strip A of Fig. 3, the pressure recordings are from 43, 48 and 53 cm. below the incisor teeth. The orifices of all three catheters are within the stomach. Inspiration gives a positive respiratory deflection when the catheter is located below the diaphragm.

In strip B the first catheter at 40 cm. is located within the lower esophageal sphincter. The resting pressure has risen to approximately 15 mm. of mercury above atmospheric pressure; moreover, swallowing evokes a relaxation of the sphinc-

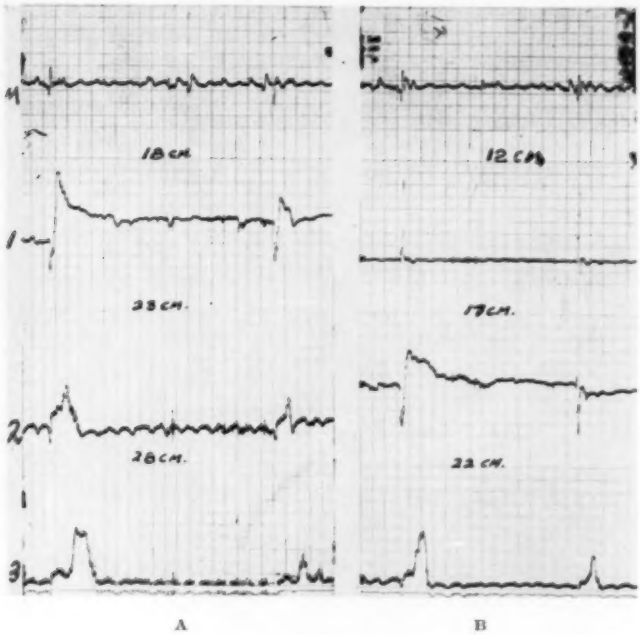

Fig. 4.
A—Catheter #1 in Cricopharyngeus, 2 and 3 in esophagus showing peristaltic waves with swallow (deflection on myogram).
B—Catheter #1 in pharynx.
" #2 in cricopharyngeus.
" #3 in esophagus.

ter followed by contraction. The two lower catheters are still located within the stomach on this strip.

In strip C the catheters are at 20, 25 and 30 cms. A typical, progressive esophageal peristalsis is demonstrated. The first catheter at 20 cm. is just below the cricopharyngeal sphincter and a small relaxation phase is already present.

Fig. 4 is a continuation of the tracing depicted in Fig. 3. In strip A the first catheter at 18 cm. has just slipped into the cricopharyngeal sphincter. There is a marked rise in resting pressure. On swallowing, a relaxation phase of at least 15 mm. of Hg. followed by a strong contraction is pres-

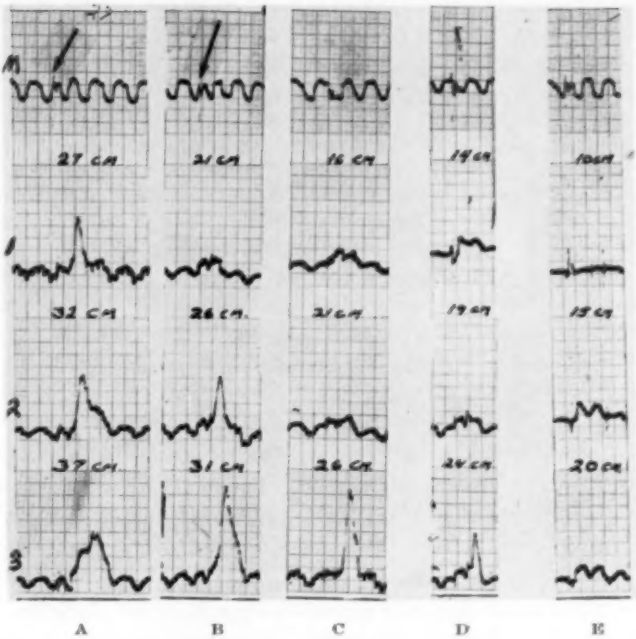

Fig. 5.

A—Normal esophageal peristalsis.

B, C, D, E show area of decreased peristaltic activity 3 to 5 cms. below cricopharyngeus, a common finding after laryngectomy.

ent. The second and third leads are still in the esophagus and a typical esophageal peristalsis can be seen.

In strip B the first catheter is within the pharynx. The resting pressure has fallen to atmospheric levels. Swallowing evokes a sharp, rapid rise and fall in pressure. The second catheter at 17 cm. is within the cricopharyngeus and again there is a rise in resting pressure and a characteristic response to swallowing. The third catheter is within the esophagus.

Fig. 5 is a tracing taken upon a 41-year-old female who had had a laryngectomy approximately three years before these studies were made. This patient had developed a good esophageal voice.

In strip A all three catheters are within the esophagus and demonstrate a normal esophageal peristalsis. In strips B and C the first catheter is at 21 and 16 cms. and the esophageal peristalses are of very low amplitude. This area of decreased activity is also reflected in strips D and E for the second and third catheters.

This hypoactive area, measuring some 3 to 5 cms. below the cricopharyngeus, was present in eight of our postoperative patients. Whether this represents a denervated segment of the esophagus or whether the lumen has been weakened by closure of the anterior wall to such an extent that normal pressures cannot be generated is difficult to determine. It is interesting to note that in spite of this relatively inactive area, the peristaltic wave passes down the remainder of the esophagus in a normal fashion.

In strip D the first catheter is located within the cricopharyngeus. There has been very little rise in resting pressure. The relaxation phase following swallowing is extremely small, and the ensuing contraction is markedly diminished.

In the final strip, the first catheter is within the pharynx. The pharyngeal response to swallowing is of low amplitude. The second catheter in this strip is within the cricopharyngeal sphincter and again demonstrates decreased activity in this area.

Fig. 6 represents the tracings taken upon a 41-year-old male who had had a laryngectomy three years prior to these studies. This patient had an excellent esophageal voice. He was reluctant to have studies made because of an active gag reflex. The irregularity of the myogram is due to gagging throughout the procedure. The second catheter became plugged during the studies, so only leads 1 and 3 are pictured. This patient is presented because he was the only patient with a good esophageal voice who demonstrated an active cricopharyngeal sphincter.

In strip A the first catheter at 25 cm. is within the esophagus and peristalses of moderate amplitude are demonstrated.

At 20 cm. the catheter has just entered the cricopharyngeal

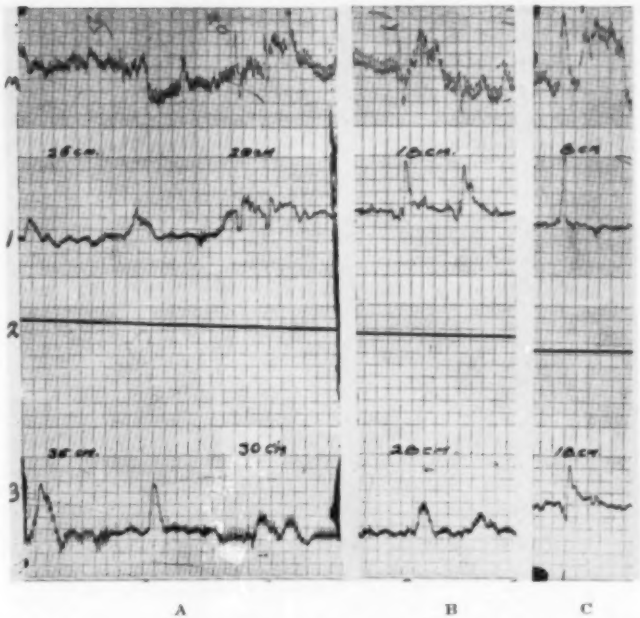

Fig. 6. Upper esophageal sphincter in patient with good esophageal voice.
A—Rise in pressure at 20 cms. level.
B—Sphincter area in tube #1 showing relaxation phase on swallow.
C—Third catheter was within sphincter area, as demonstrated by rise in baseline.

sphincter and a rise in resting pressure is noted. A relaxation phase of small degree is present. At 18 cm. the catheter is well within the sphincter. The high level of resting pressure is maintained. A very small relaxation phase and a good contractile element are present after swallowing. In the final strip, the first catheter at 8 cm. is within the pharynx. An active pharyngeal contraction occurs in response to swallowing. The resting pressure has dropped somewhat, but not to normal levels. The third catheter at this level is within the cricopharyngeus.

A number of our laryngectomized patients developed a sphincter postoperatively as evidenced by a rise in resting

pressure. Some of these, like this patient, evoked a fairly normal contraction following swallowing. The relaxation phase, however, was either very weak, or entirely absent. Never was the relaxation phase normal; moreover, if a rise in resting pressure occurred, the area involved appeared to include the upper esophagus, the cricopharyngeus and the

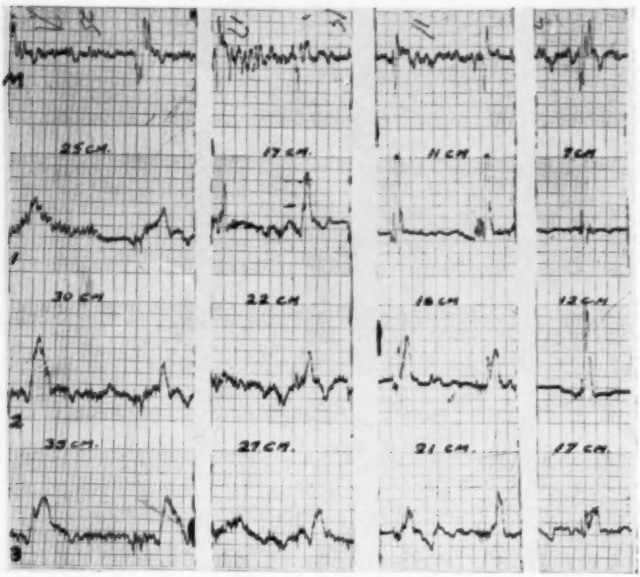

Fig. 7. No rise in pressure at upper end of esophagus, in patient with excellent esophageal speech.

lower pharynx. These three areas appeared to contract as a whole.

Fig. 7 is the tracing of a 60-year-old male who had had a laryngectomy five years prior to these studies. He had an excellent esophageal voice. In the first strip the three catheters at 25, 30 and 35 cms. are in the esophagus. Normal esophageal peristalses are seen. Although readings were

made at various levels up to 7 cm. (the posterior tongue), there is no significant pressure variation between the upper esophagus, cricopharyngeus and pharynx. A sharp contractile spike replaces the broad esophageal peristalsis in the uppermost areas in response to swallowing, but no definite sphincter area is demonstrated.

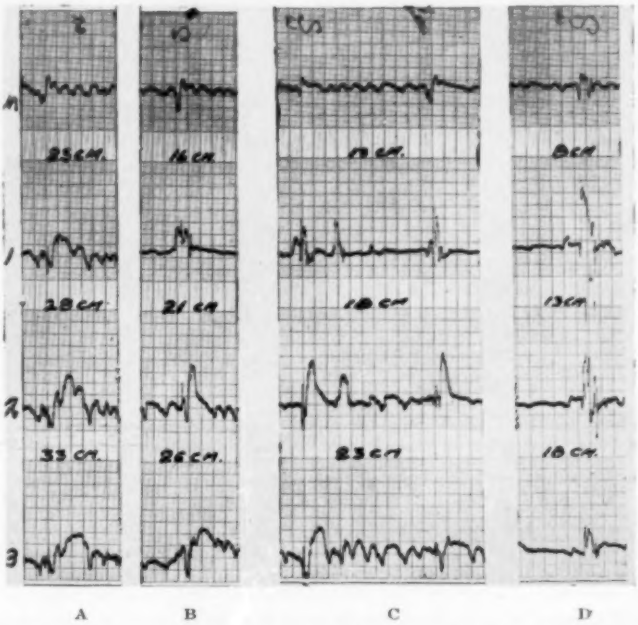

Fig. 8. No pressure rise at upper end of esophagus, in patient with excellent esophageal speech on both inspiration and expiration.

Fig. 8 is the tracing of a 71-year-old male who had had a laryngectomy five years prior to these studies. This patient had the best voice of our series. Although there is no evidence of a sphincter in these studies, this patient was able to speak upon both inspiration and expiration; and rather than swallow air as the usual laryngectomized person, he was able to

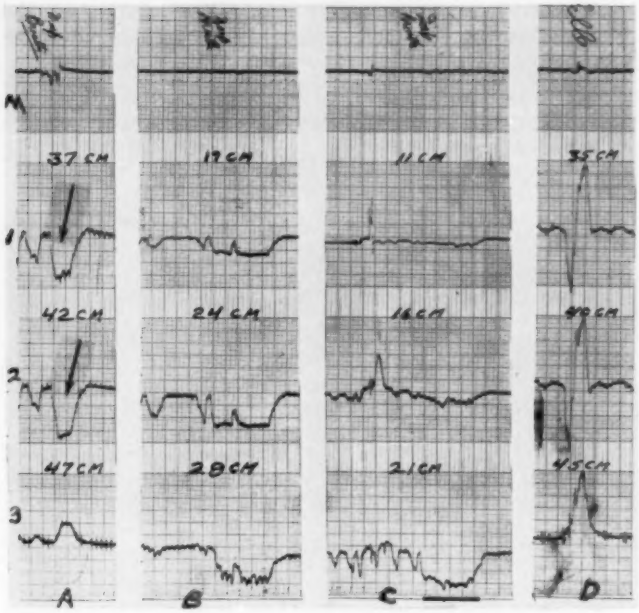

Fig. 9. Effect of deep inspiration on intraesophageal pressure in patient with no demonstrable sphincter in upper esophagus. (See Fig. 8.)
A—Inspiration produces a sharp drop in intraesophageal pressure, showing that air is not aspirated into esophagus.
D—Sharp drop and rise on rapid inspiration, during which patient speaks.

aspirate air into his esophagus at will; however, on normal inspiration no air passed into the esophagus.

Studies were made upon this subject again at a later date, and again no upper sphincter was demonstrated. Portions of this tracing are shown in Fig. 9. In the strip A the upper two catheters are at 37 and 42 cms. and are located within the esophagus. The lower catheter is located within the stomach. Upon taking a deep inspiration, there was a fall in pressure in the two catheters above the diaphragm and a rise in pressure in the catheter located in the stomach. This was interpreted as showing that air was not aspirated into the esophagus on deep inspiration. If such were true, the pres-

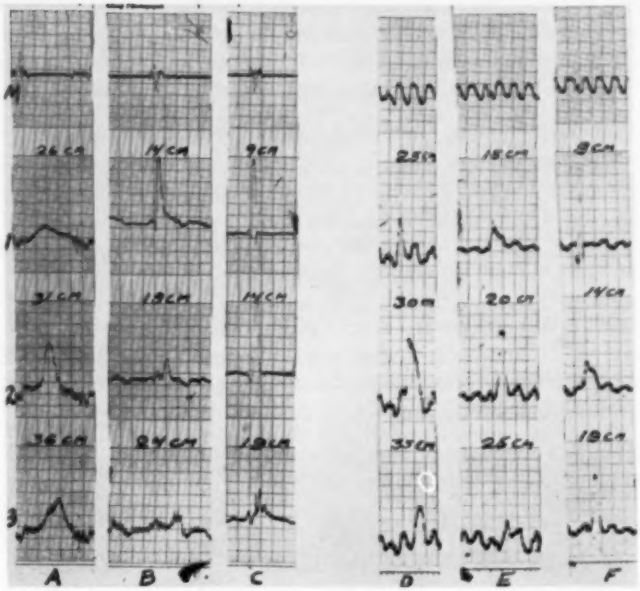

Fig. 10.

A, B, C—Excellent upper esophageal sphincter, in patient with no esophageal speech.

D, E, F—No upper esophageal sphincter, in patient with no esophageal speech.

sure within the esophagus would remain constant at atmospheric levels.

In strip B the first catheter is at 19 cm. and is located in the lower portion of the cricopharyngeus. Deep inspiration decreases the resting pressure only a small amount at this level while, lower down, the intraesophageal pressure fell as before.

In strip C the upper catheter is in the pharynx and the second catheter in the upper end of the cricopharyngeus; neither appears to be affected by deep inspiration.

The final strip was taken while the patient was vocalizing. Upon saying "Hello," the patient inspired rapidly. There was

a rapid, brief fall in intra-esophageal pressure as shown by the first two catheters. This was followed by a brief, sharp rise in pressure above resting levels. The third catheter, located within the stomach, recorded only a rise in pressure.

It would appear then that although this subject demonstrated no cricopharyngeal sphincter, air did not flux into the esophagus upon normal respirations or even upon deep breathing; however, the patient was able to aspirate air into the esophagus at will when he desired to speak (see strip D). The exact method by which the patient accomplished this is not known, but it was noted that upon speaking the patient inspired and expired in short blasts, so that the degree of negativity within the esophagus probably assumed greater proportions than that produced by normal breathing or even by taking a deep breath.

The final slide is of two patients (see Fig. 10). The first three strips are from a patient who is 18 months postoperative. The last three are of a patient over five years postoperative. In the first instance the patient has an excellent esophageal sphincter; in the second, little or no sphincter element is present. These two patients had one element in common: neither was able to speak. This serves to illustrate that whether or not a sphincter is present, other factors are important in developing an acceptable voice after laryngectomy.

DISCUSSION.

The use of transducers and a suitable recording apparatus in our hands has proven a useful tool for the study of the pressure dynamics of the cricopharyngeal sphincter. The sphincter exists as a distinct physiological entity and is easily identified by this method.

After studying a number of normal subjects we were able to recognize several major phases of sphincter activity. The sphincter is in a state of constant tonus during the resting phase. The pressure within the sphincter is 10 mm. or more of mercury above intra-esophageal pressure. This high pressure zone extends for a distance of 3 to 4 cm. and has been

described by previous investigators using methods similar to ours. Presumably the upper esophagus, and perhaps even the lower pharynx, in addition to the cricopharyngeus muscle, contribute to this sphincter mechanism.

The act of swallowing is preceded by a phase of relaxation of the entire sphincter with an abrupt fall in pressure to intra-esophageal levels. The sphincter then rapidly contracts, with a rise in pressure to 10 or more millimeters of mercury over resting levels. The act of swallowing then being completed, the pressure falls to previous resting levels.

We have found that one or more of these pressure phases are either absent or greatly altered in the laryngectomized patient. The resting pressure in four of our operated patients never exceeded intra-esophageal levels. In 12 patients, the increase in pressure was less than 5 millimeters of mercury. In performing a laryngectomy, we have always attempted to reconstruct a sphincter by approximating the cut ends of the cricopharyngeus muscle. It is obvious from our studies that we were never completely successful.

Since the reconstructed sphincters in all of our operated patients appeared to be lacking in tone, it is not surprising that the phase of relaxation that occurs at the onset of swallowing was either absent or greatly diminished. Even those patients who developed a significant resting pressure postoperatively showed a disturbance of this phase of swallowing. It cannot be assumed that the lack of tone in the postoperative sphincter and its failure to relax with swallowing is due to a disturbance of innervation or to incomplete closure of the sphincter at the time of surgery, since the final phase of swallowing, sphincter contraction, was always present and almost always of normal amplitude. One would assume that this phase of swallowing would suffer most from incomplete closure of the muscular ring or from an altered innervation.

The segment of esophagus immediately below the cricopharyngeus showed an altered pattern in six patients. The amplitude of the esophageal peristalsis was markedly depressed in these subjects. This type of disturbance would be

compatible with incomplete closure of the muscular ring or with a disturbance of the innervation of this portion of the esophagus. Of more importance was the fact that once the peristaltic wave transversed this portion of the esophagus, it passed down the remainder of the esophagus in a normal fashion.

The most significant finding in this study was that the cricopharyngeal sphincter alone is neither necessary for the production of esophageal speech nor responsible for the prevention of esophageal respiration. The patient illustrated in Fig. 8 was one of several with a good esophageal voice who had no evidence of a sphincter. On normal or even deep inspiration, air did not enter the esophagus; however, the patient was able to insufflate air by taking a sudden, forced inspiration, and when this occurred, the pressure in the esophagus immediately fell to sub-atmospheric levels, and air rushed into the esophagus.

SUMMARY.

The normal sphincter closure of the upper end of the esophagus is usually absent after laryngectomy. In spite of this there is no difficulty in swallowing nor is air sucked into the esophagus on ordinary or even deep inspiration. The prevention of esophageal respiration is not an important function of the upper sphincter mechanism. Esophageal speech does not depend upon a functioning sphincter at the upper end of the esophagus. Our best speaker had no demonstrable sphincter and could speak on both inspiration and expiration.

The peristaltic activity of the lower esophagus and the function of the lower esophageal sphincter are not altered by disturbances of function of the upper esophagus or of the cricopharyngeal sphincter.

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A TECHNIQUE OF TYMPANOPLASTY.

Preservation of the Bony Canal Wall. Use of Vein Grafts.*

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HISTORICAL NOTE.

Attempts to close perforations of the tympanic membrane have been made for several centuries and by a variety of techniques. The first attempts were made with prostheses. Banzer[1] in 1640, used membrane from a pig's bladder stretched over an ivory tube. He did not succeed, and his successors, who used other devices, had no better luck. Hope dies hard, however, and as late as 1951, Pohlman[2] applied the prosthetic principle again, with the use of a rubber-like plastic, Korogel.

The first attempts to close a perforated tympanum with skin seem to have been made by Berthold,[3] in 1878. He had a single ten-year success, but all his other operations were followed by either indifferent results or absolute failure. Other otologic surgeons who also used skin for this purpose had no better results, and the technique was dropped until shortly after World War II. Then it was revived, independently, by Zöllner[4] in Freiburg and by Wullstein[5] in Wurzburg, after they began to see many ears that had been damaged during the wartime bombing of German cities. When they began their work there were a number of auspicious circumstances, including Lempert's development of temporal bone surgery, a greatly increased knowledge of auditory physiology,

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and the availability of magnifying operating otoscopes of remarkably increased power.

In 1953 House[6] reported four cases in which he had used skin to bridge the ruptured tympanum. His first attempt, made seven years earlier, had not been successful, but he had good results in the other three cases. Interest in this technique has grown successively in the United States, and there are now numerous reports on it in the literature.

SURVIVAL OF SKIN GRAFTS OF THE TYMPANUM.

The survival of the graft is the crux of the problem in tympanoplasty, no matter what tissue is used for this purpose. The mechanism by which grafts take in the ear differs fundamentally from the mechanism by which they take elsewhere in the body. This might be expected, since anatomic conditions in the ear are not duplicated elsewhere in the body.

Garré's[7] classic observations on grafts, made in 1888, have been confirmed many times. In a study of the histologic changes in human skin grafts made from five hours to two-and-a-half years after their application, he found *1.* that invasion of the graft by the capillary buds of the host tissue began on the third or fourth day; *2.* by this time, most of the vessels in the graft itself had become obliterated. His opinion was that grafts were able to survive the interim between the application of the graft and the invasion of the host vessels by virtue of what he termed "plasmotic circulation." By this he meant the absorption by the graft of plasma and cellular components from the host.

Garré's explanation might be acceptable for grafts placed directly on tissue containing a bed of blood vessels. It does not offer a rational explanation for the success of grafts used in tympanoplastic procedures in which the graft must bridge an air-containing cavity, that is, the middle ear. The only bed in this type of graft is peripheral; the central portion is not in contact with any tissue or any blood supply.

In 1956, almost 70 years after Garré's studies, Converse and Rapaport[8] studied the process of vascularization in 15

skin grafts. Their observations, it must be remembered, were made by stereomicroscopy and oil-immersion examinations, refined techniques that were not available to their predecessors. Their findings were as follows:

1. No vessels at all were demonstrable during the first 24 hours after the application of the graft.

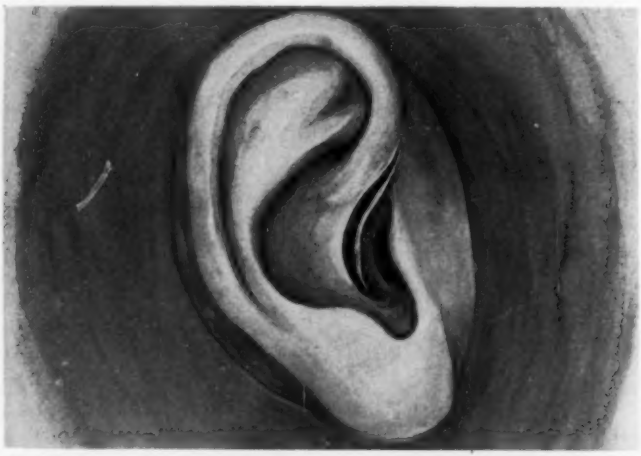

Fig. 1. Technique of tympanoplasty with venous graft. Endoaural incision, which must be near cartilage, to facilitate removal of sutures. This precaution is especially important in young children. Before the incision, the mucosa of the inner surface, and the epithelium of the outer surface, of the drumhead are stripped from the remnants of tissue left after perforation.

2. During the second 24 hours, vessels were observed that gradually became dilated and filled with static blood.

3. By the third or fourth day, a sluggish flow of blood was always evident in these vessels. The flow gradually increased, and by the fifth or sixth day vascularization was generalized.

4. Over the next several days the vessels increased in number, and by the tenth day, the vascular pattern of the graft was essentially the same as that of the surrounding tissues. By the twelfth day, all the grafts had, grossly, the same color and appearance as the surrounding skin of the host.

Fig. 2. Dissection of membranous wall from spine of Henle and tympanosquamous suture. The subcutaneous tissue of the wall of the membranous canal has already been removed. The mastoid is approached in the epitympanic area by means of a cutting burr.

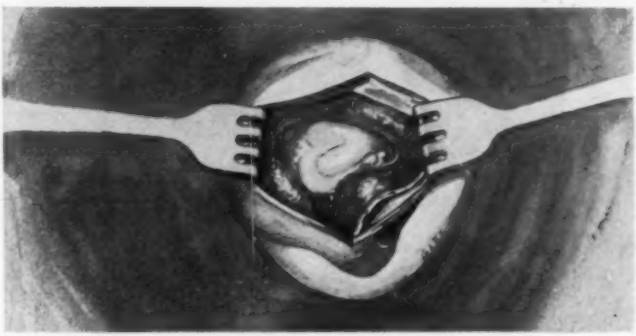

Fig. 3. Mastoid process freed of all cells. It is important that they be completely removed, to permit easy visualization of the malleo-incudal junction.

These are interesting observations, but, as already intimated, they are of no help in explaining why a skin graft can survive when it is placed over a perforation of the tympanum. The theory that some type of circulation exists and provides nourishment for the central portion, over the air-containing cavity, remains to be proved. Other speculations are no more acceptable. To date, it is still not possible to

explain why a graft of this kind takes, or why the results of some grafts are excellent while in other cases, in which the circumstances seem precisely the same and in which the procedure has been carried out in exactly the same fashion, the end-result is failure. We shall not understand the variability of results until we learn why suspended skin grafts survive for the first few days after their application.

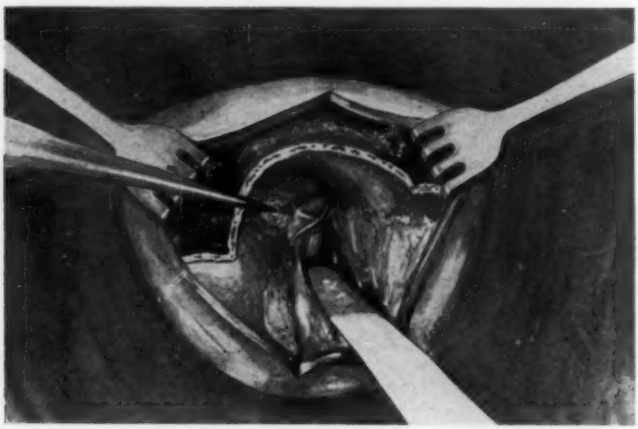

Fig. 4. Visualized middle ear. Visualization has been facilitated by thinning of the bony canal wall from the medial aspect of the middle ear.

EXPERIMENTAL STUDIES ON SKIN GRAFTS.

Our own attempts to solve the mystery of why grafts do and do not take in tympanoplasties began with clinical studies. It took only a few clinical experiments to prove to us that, desirable as it might be to make such studies, they were simply not feasible. The necessary procedures were often extremely painful. The residual scarring was highly undesirable. Finally, and most important, there was real danger that these experiments, if they were continued, might jeopardize the end-results of tympanoplasty. They were, therefore, discontinued, and experimental studies were undertaken.

For our purposes, pigs seemed the preferred test animals,

and pigs bred by the Hormel Institute at the University of Minnesota in Austin, Minn., seemed the most desirable breed. Except for the gorilla, whose experimental use is obviously impractical, the skin of the pig most closely resembles the skin of the human subject. The epidermis is about the same as that of the human subject or perhaps is slightly more delicate. The distribution of hair and pigment is fairly uniform, as in the human. The corium, however, is slightly thicker than human corium. Pigs bred at the Hormel In-

Fig. 5. Use of cutting burr to remove overhang of bony canal and freeing of annulus, after which complete visualization of middle ear is possible.

stitute also have a practical advantage: their care and experimental use are simplified by their small size; even at full growth they are not more than a third the size and weight of ordinary swine of parallel age.

The experiments undertaken in these pigs duplicated clinical perforations of the tympanum. After the perforations had been created, skin grafts were suspended across them, and studies of the survival of the grafts were made. Our observations were limited because of numerous difficulties which arose. One of them was the death of one of the animals,

which provided a barbecue for the animal house attendants but threw no light on tympanic grafts.

Such observations as we were able to make on these animals, however, convinced us that the theory of immediate mouth-to-mouth establishment of a host-graft circulation is not tenable. We also found nothing to support Garré's[7] theory of a "plasmotic circulation"; in fact, our personal observa-

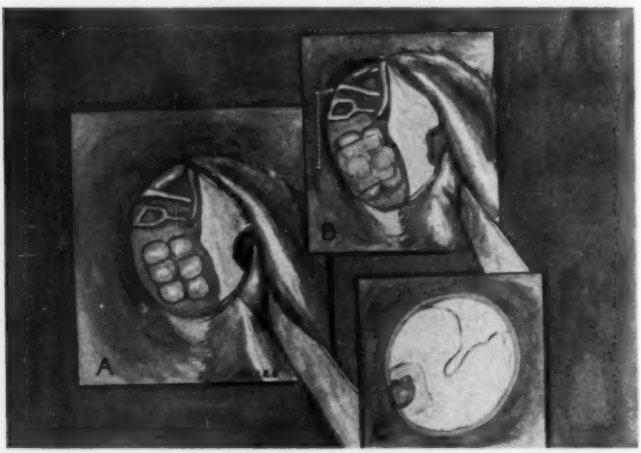

Fig. 6. Closure of perforation in tympanum. A. Application of gelfoam soaked in haluronidase solution and antibiotic (of choice) in middle ear, to provide support for vein graft. B. Placement of vein graft over gelfoam. C. Restitution of fibrous remnant of drumhead to its former position.

tions refute both of these theories; on occasion, we have refrigerated grafts for six days or longer and still had perfectly satisfactory results when we used them in temporal bone surgery.

Our limited experimental observations, supplemented by a considerable clinical experience, have brought us to the conclusion that the survival of grafts in perforations of the tympanum depends upon the establishment of an adequate vascular system. Such a system is established by the growth

of endothelial buds that arise from the blood vessels in the peripheral bed, extend out into the graft, and join in continuity with the blood vessels of the graft. Most investigators agree with the fundamental concept of an adequate vascular system, but they believe that this process occurs between the third and tenth days after operation. Our observations suggest that in grafts that take, this circulatory union occurs very soon after the graft is applied. In those that do not

Fig. 7. Repair of very large perforation of tympanum by technique just described. It is essential that the drum be stripped of all remnants and that no scar tissue is left in situ.

take, the union either does not occur at all or does not occur early enough to maintain the viability of the graft.

If these observations and conclusions are valid, as we believe they are, several possible reasons would explain failures in plastic surgery on the tympanum, including:

1. Trauma to the graft while it is being obtained from the donor site, principally from thinning it with scissors. We abandoned this technique some time ago and now use a knife for this purpose.

2. The presence of pleats or folds in the graft, which prevents making good contact with its bed and further results in paucity of the blood supply.

3. Improper packing of the ear, which produces such pressure that the endothelial buds cannot develop in the graft.

4. Infection of the graft.

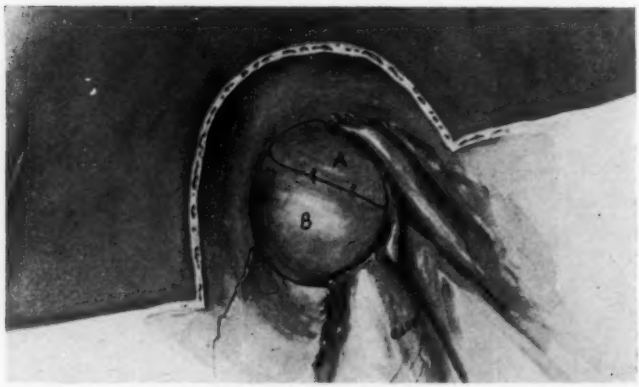

Fig. 8. Application of vein graft in cavum minor technique (Wullstein's type III operation), used when columellar effect is necessary. The epitympanic area is closed off by application of the vein (a) with the adventitia against the bone and (b) against the hypotympanum with the adventitia external. If approximation is not complete, the vein grafts must be sutured together.

Since all of these factors are controllable, it seemed to us, once we had become aware of them, that the problem of tympanic grafts would be solved; it was not! All of the difficulties listed occurred early, within ten days of operation, and we no longer lost grafts at this time. We found, however, that we were losing them months later, after we had considered the tympanoplasty a success.

Analysis of our material showed several reasons for the late loss of tympanic grafts:

1. Ingrowth of the epidermis through the cut appendage (glands and hair follicles), through holes of infinitesimal size made during the process of thinning the graft.

2. Resultant cholesteatoma-formation, with resultant impairment of hearing.

3. Infection or circumscribed cellulitis of the submucosal layer of the graft, with resultant perforations and cholesteatoma-formation.

4. The presence of rugae or folds in the suspended portion of the graft. These were invariably present, no matter how carefully the grafts were placed. Since the skin involved in

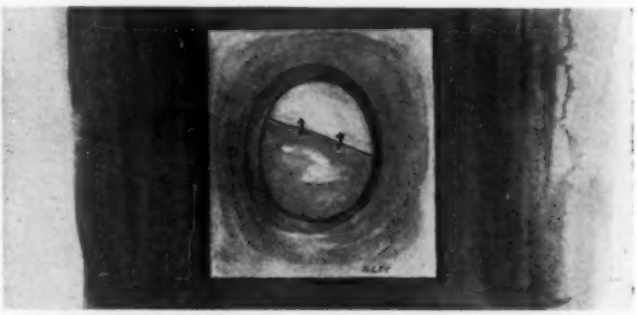

Fig. 9. Coverage of portion of vein applied to bony canal by skin of ear canal, thus providing for epidermis to grow over the vein graft. For this technique to be successful, the vein graft must be placed well up on the wall of the canal.

the rugae desquamated, inclusion cysts formed, and the graft was eventually destroyed.

VENOUS GRAFTS IN TYMPANOPLASTIC SURGERY.

Because of our disappointing results with skin grafts, particularly their late loss, we began to search for a more suitable technique for repair of perforations of the tympanum. The results were equally discouraging whether fascia, amniotic membrane or oral mucosa was used to line the middle ear after its mucosa had been removed.

In October, 1959, in discussing a paper by Withers and his associates, on the clinical implications and effects of burying epithelium experimentally in the middle ear, I made a brief

report on a single case in which a vein graft had been used in a primary tympanoplasty.[9] The anatomic and functional results in this case were excellent.

In March, 1960, Tabb[10] reported 20 cases in which vein grafts had been used in myringoplasties to repair large perforations of the tympanic membrane, particularly perforations of long standing. The results in these 20 cases, and in the

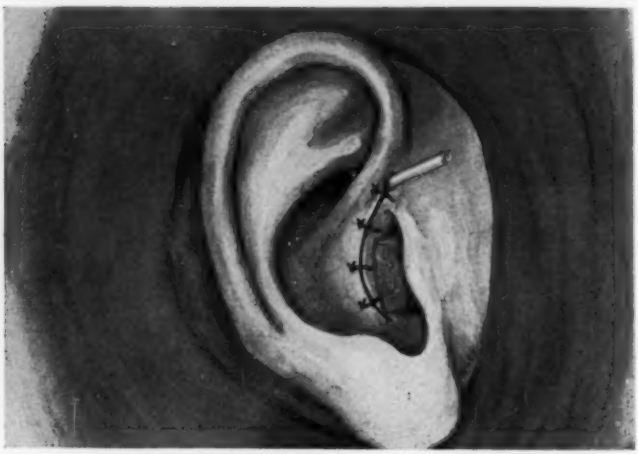

Fig. 10. Polyethylene tube inserted down to antrum and sutured in incision, which has been closed with interrupted sutures. A small piece of surgical rayon has been placed over the graft, and gelfoam soaked in antibiotic-haluronidase solution has been applied over the rayon. Finally, a sea-foam sponge has been inserted just firmly enough to hold the membranous canal against the enlarged bony canal. Pressure must be avoided.

47 cases in which the technique has since been used, were so generally good that we have extended the use of vein grafts to all tympanoplasty surgery.

TECHNIQUE OF TYMPANOPLASTY WITH VEIN GRAFTS.

The technique that we employ in vein grafts for tympanoplasty (see Figs. 1-10) is much the same as the technique described by Myers and Schlosser[11] earlier this year, except

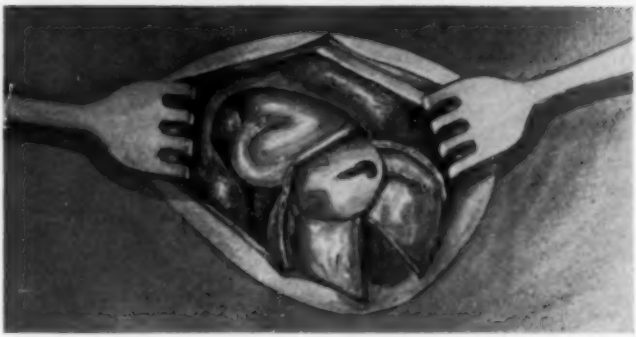

Fig. 11. Complete removal of necrotic mucosa of middle ear and mastoid bone, with creation of pedicle flaps of skin of aural canal.

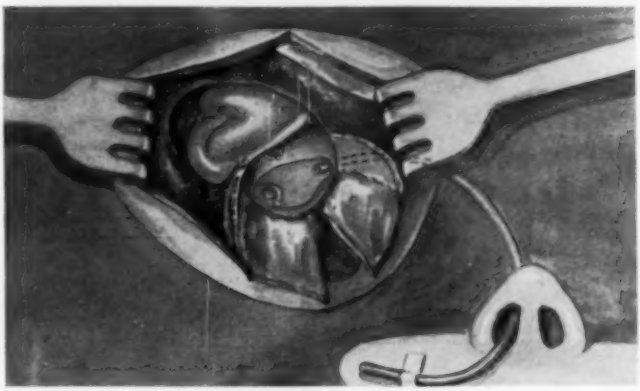

Fig. 12. Technique of preservation of orifice of Eustachian tube when mucosa must be removed from middle ear. The vein graft is placed in contact with the mucosa of the Eustachian tube and up to the round window niche, with the adventitia toward the bone. The polyethylene tube inserted through the Eustachian tube is brought out through the nose.

that we employ an endaural approach. The great advantage of this technique is that the posterior wall of the bony canal is preserved, and there is, therefore, no postoperative mastoid cavity, even though one was created at operation to eradicate the disease. This is a great advantage. If the bony wall is taken down, the residual cavity into the mastoid requires life-

time care, which is particularly unfortunate if the patient is a child. The technique illustrated eliminates this situation. The surgical cavity is allowed to heal naturally. The drain, which is kept in place from four to six days, prevents infection and permits irrigation of the cavity with appropriate antibiotic solutions if infection does occur.

Regardless of the pathologic process in the tympanum and its extent, it is now our practice to use vein grafts in tympano-

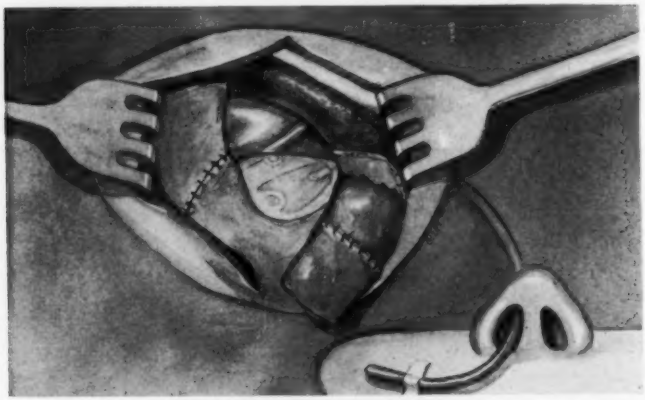

Fig. 13. Formation of cavum minor hypotympanum by application of vein graft with intima against stapes. Three-fourths or full-thickness skin, which will eventually be covered with epidermis, is used to cover the remainder of the middle ear and the mastoid. It must be carefully placed over the margins of the vein graft.

plasty if the bony wall of the canal can be kept intact. If the ossicular chain is so involved in the antecedent infection that the malleus and incus must be removed, then one must rely upon a columellar effect. In these operations, the vein graft is dropped directly onto the stapes, after which the epitympanic space is closed off, the adventitia of the vein graft being in contact with the bone. In these circumstances it is sometimes necessary to use two pieces of vein, which sometimes, though not always, must be sutured together to ensure satisfactory apposition (see Fig. 8).

When it is not possible to preserve the integrity of the bony canal and a mastoid cavity cannot be avoided, another technique must be employed (see Figs. 11-14). This technique is employed, however, only if it is certain that the control of the patient will be such that adequate postoperative care can be assured. If there is any doubt on this point, a plastic type of operation, using muscles, must be carried out by much the same technique Rambo[12] has described for primary closure of a radical mastoid wound.

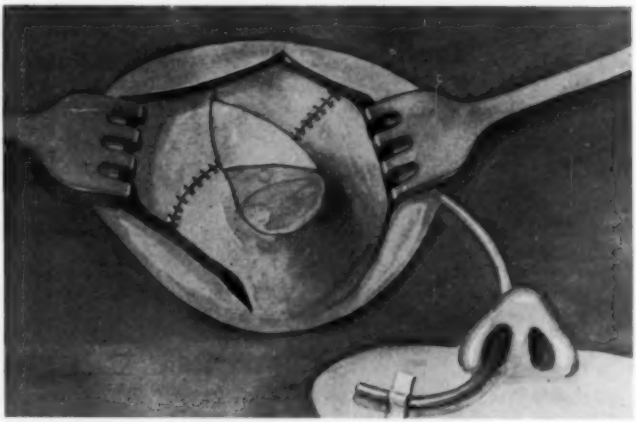

Fig. 14. Suture of pedicle flaps to skin graft and coverage of epitympanic area with skin.

COMMENT.

When a vein graft is used in a perforation of the tympanum in which remnants of membrane are still present, the graft probably serves as a tissue splint for them. When such ears are entirely healed, it is impossible to tell, even under high magnification, where the graft has been applied. This explanation does not seem to cover the success of grafts in ears where no such remnants are present. In the technique that we employ, the edges of the vein graft are fitted to the walls of the aural canal, and the skin of the canal is brought down

over them like a cuff. Our theory is that in these cases, the epidermis of the skin of the canal grows out to cover the vein graft, at first imperfectly and later completely.

At the present time, whenever we must replace the mucosa of the middle ear, it is our practice to use a vein graft. We also use venous tissue, for the suspended portion of the necessary graft, in all tympanoplasties. Whether this technique will provide the answer for all the difficulties encountered with full-thickness skin grafts it is too early to say, but the outlook seems promising. Certainly during the year in which we have performed tympanoplasty with vein grafts, in 63 cases to date, this method has given more gratifying results than we have obtained with any other technique.

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EXPERIMENTAL FINDINGS FOLLOWING THE STAPES REPLACEMENT PROCEDURE.*

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There has been a renewal of interest and improvement of techniques since the revival of stapes surgery by Rosen[1] in 1952. Considerable interest has been shown in the reactions of middle and inner ear structures to trauma and the introduction of substances commonly used in the different procedures. Serially sectioned and stained human temporal bones in which these procedures have been performed are usually not available. For this reason several investigators have reported their findings after similar procedures on experimental animals. The results obtained by fracture of the stapes in rabbits have been described by Altmann and Basek.[2] Trauma to the middle ear ossicles and oval window region and tissue reactions with reconstruction of the latter have been reported by Bellucci and Wolff.[3,4] Changes following fracture of the stapes in cats were reported by Singleton and Schuknecht[5] and the acoustic trauma experimentally induced by stapes manipulation, by Schuknecht and Tonndorf.[6] The formation of mucoperiosteal membranes in the obliteration of defects in the oval window after fractures and fenestrations of the stapes footplate were described by Schuknecht and Oleksiuk.[7] The results obtained by various tissue grafts over the oval window after stapedectomy were reported by Schuknecht, McGee and Coleman.[8]

The present study is a further attempt to observe the reactions on the part of the middle and inner ears of cats, to stapedectomy; the subsequent fate of the gelfoam insert over the oval window, and the polyethylene prosthesis.

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METHODS.

Twenty-two ears of cats were used for stapedectomy. Veterinary pentobarbital (Nembutal Sodium) was injected intraperitoneally for anesthesia. In each, half of the head and neck was shaved. The skin was scrubbed with septosol soap, then rinsed with 1:1000 benzalkonium chloride solution (Zepharin). After drying, the area was finally painted with tincture of iodine and rinsed with 70 per cent alcohol. All instruments and drapes were autoclaved except the drill bit and headpiece which were sterilized in benzalkonium chloride (Zepharin). Strict aseptic technique was adhered to throughout. After draping the animal, the middle ear was exposed through a suprameatal atticotomy by way of a postauricular incision. A good view of the incudostapedial joint, stapes and footplate could be obtained after retraction of the facial nerve. The incudostapedial joint was disarticulated with a House joint knife, the stapedius tendon cut and the stapes gently tipped to the side and lifted out. In two animals the footplate was inadvertantly depressed. A compressed quarter-thickness 2×2 mm. gelfoam pad was placed over the oval window and a 1.5 mm. No. 50 polyethylene strut was placed with the tapered end resting on the gelfoam and the other around the lenticular process of the incus. In the earlier animals the soft tissues were closed by primary suture. Later a gelfilm disc was placed over the atticotomy defect in an attempt to prevent fibrous invasion of the overlying soft tissues into the middle ear. The tympanic membrane and external auditory canal were not disturbed. Great care was taken to keep the middle ear free of blood and bone debris. This was accomplished through Ringer's lactate irrigating solution and fine suction. After completion of the surgical procedure 600,000 units of procaine penicillin was administered intramuscularly.

These animals were sacrificed at varying intervals from 48 hours to four months. The temporal bones were removed immediately and immersed in Heidenhain-Susa fixative. Subsequently they were decalcified in 5 per cent hydrochloric acid, embedded in celloidin, sectioned and stained in hemotoxylin-eosin.

RESULTS.

It was possible to classify the microscopic findings for this series of temporal bones into three categories on the basis of presence and degree of pathological changes:

Group I. No inner or middle ear changes demonstrable postoperatively.

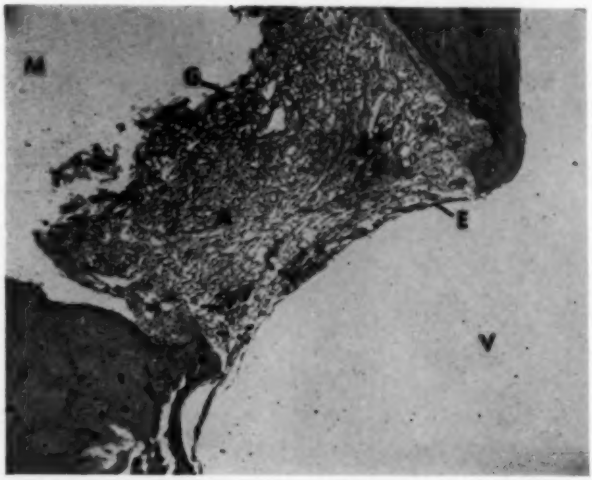


Fig. 1. Replacement of most of the gelfoam insert in the oval window by loose areolar tissue (A). A thin endothelial-like membrane (E) is apparent on the vestibular surface. Small pieces of gelfoam (G) still persist. There is no encroachment of the connective tissue upon the vestibule (V). Two months after stapedectomy. (34X.)

Group II. Fibrosis in the middle ear but no alterations of the inner ear structures.

Group III. Inflammatory disease present, either otitis media, labyrinthitis or both.

Group I.

Of the 22 animals operated, ten (45 per cent)* failed to

*All percentages expressed are in reference to the entire series of 22 operated ears.

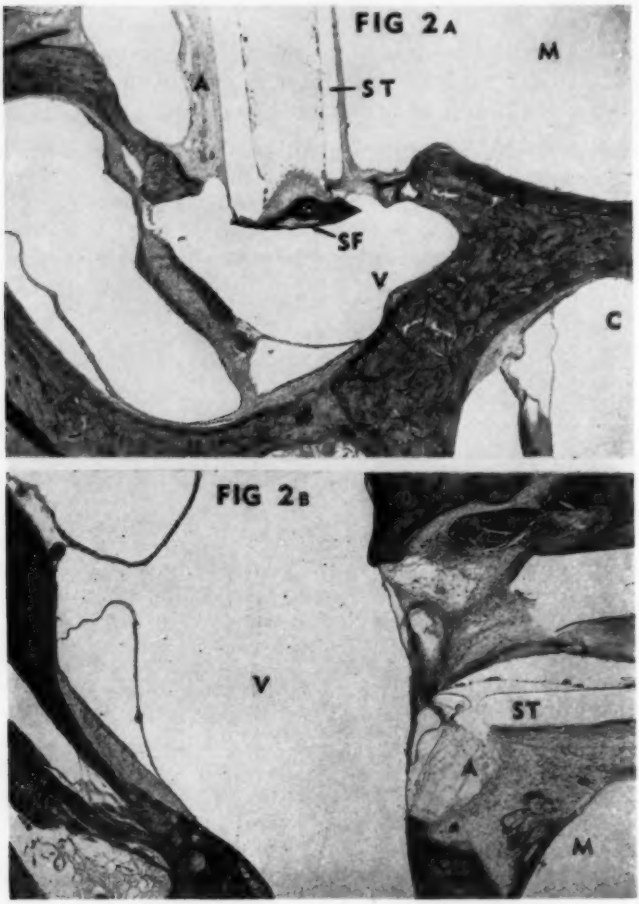


Fig. 2-a. The footplate of a stapes (SF) depressed into the vestibule (V) with fibrous tissue connections to the oval window margin and polyethylene strut (ST). The latter is surrounded by mucosa and loose areolar tissue (A) and contains multinucleated giant cells in the lumen. The gelfoam inserted between the stapes footplate and the strut has been replaced by fibrous connective tissue, and some new bone proliferation is apparent on the middle ear side (M) of the footplate. There are no apparent changes in the vestibule (V) or cochlea (C). Four months after the surgical procedure. (25X.)

Fig. 2-b. The reaction about the strut is similar to that shown in Fig. 2-a. The gelfoam insert has been replaced by loose areolar tissue which is covered on its vestibular surface by a thin endothelial-like membrane continuous with the lining of the vestibule. Stapes replacement operation three months previously. (28X.)

show changes in the vestibule, cochlea, spiral ganglion, eighth nerve, membranous labyrinth or middle ear. This group varied chronologically from 72 hours to four months following surgery. Changes were encountered in the organ of Corti which could be explained on the basis of autolysis and fixation artefacts in several animals that died overnight of an epidemic diarrhea. Three (13 per cent) of these ten animals did have delicate areolar strands in the region of the long process of the incus and around the polyethylene strut. They extended down to the fibrous membrane over the oval window but did not appear sufficiently numerous or dense to limit movement or function of the ossicular chain. No inflammatory exudate was present in the middle ears, and the bony atticotomy defects were sealed over with a dense fibrous scar.

Particular attention was given to the delicate structures in the cisterna perilymphatica and in the cochlea, in sections for this group that had been allowed to survive from 72 hours through four months. The saccule and utricle did not show evidence of trauma, grossly, and microscopically, receptor cells of the maculae appeared normal (see Figs. 2-a and 2-b). The crista ampullaris and semicircular canals likewise appeared to be intact and without changes. Careful observations were made of the condition of the end organs of hearing in the cochlea since damage has been reported to these structures subsequent to stapes manipulations and replacement. The specimens studied represented postoperative material of three days, four days, one week, two weeks, two, three and four months respectively. The cochlea was observed throughout its extent; but attention was especially directed to the lower part of the basal turn, since the maximal effects of trauma due to stapes surgery might be concentrated in this region. No interruptions or alterations of the basilar, tectorial or Reissner's membrane were observed. The spiral ligament appeared unaltered. No change was seen in the limbus spiralis except for the loss of connective tissue cells in several animals (see Fig. 4-b). The organ of Corti rested in its normal position upon the basilar membrane, and no change or disappearance of the hair cells or supporting cells was seen. The tunnel of Corti was intact (see Fig. 4-a).

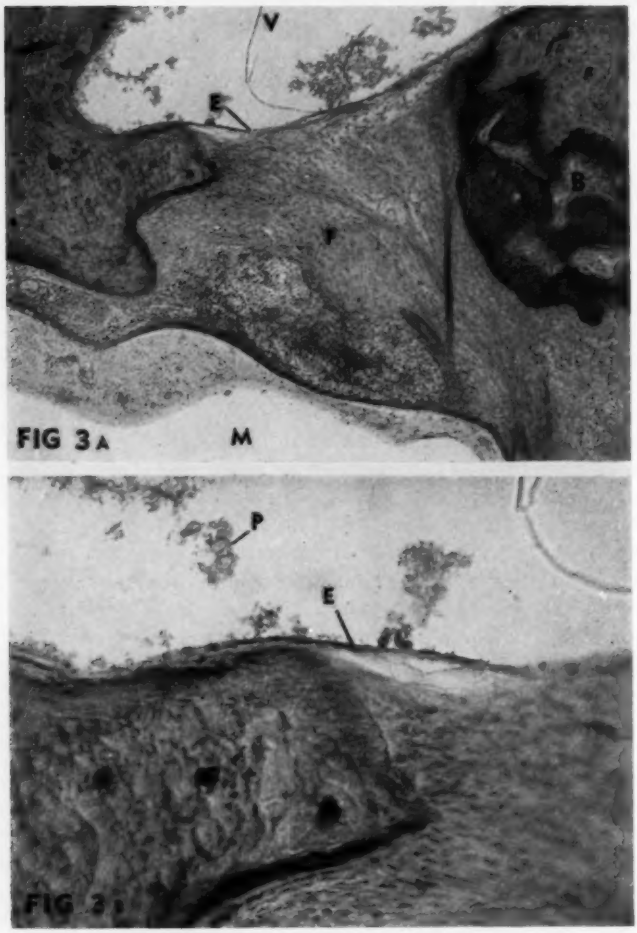


Fig. 3-a. Seven weeks following a stapes replacement procedure. The strut has slipped to lie free in the middle ear cavity. Fibrous tissue (F) has completely replaced the gelfoam, and there is some new bone (B) on one margin and middle ear surface of the oval window. A thin endothelial-like lining (E) covers the fibrous tissue on the vestibular surface and middle ear surface is covered by a well developed mucosa. (60X.)

Fig. 3-b. A higher power view of the same oval window margin seen in Fig. 3-a. The continuity of the endothelial-like membrane (E) with that of the vestibular lining is apparent. The precipitate (P) in the vestibule is acellular and is probably a fixation artefact. (175X.)

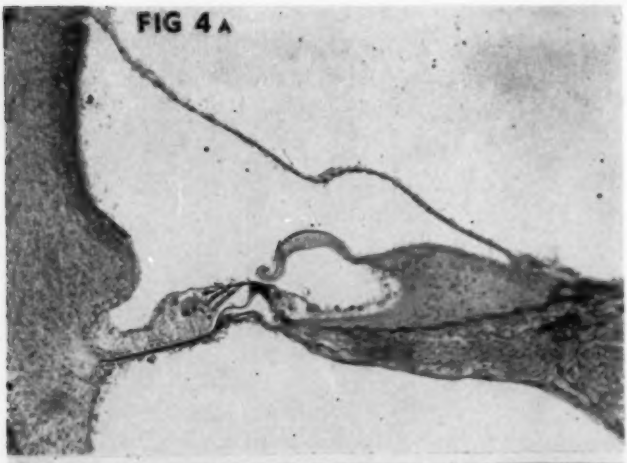
Group II.

Six (27 per cent) of the series varying from five days to two months following surgery, showed dense fibrous tissue limited to the posterior portion of the middle ear (see Fig. 5-a); it also surrounded the incus and the polyethylene prosthesis. It seemed peculiar that it did not involve the malleus or extend further forward into the middle ear. This was a dense connective tissue and in every case could be seen invading directly through the bony atticotomy defect. Certainly it was sufficient to restrict movements of the ossicular chain. The dense fibrosis was confluent with the middle ear side of the fibrous membrane spanning the oval window. In no case did it extend into the vestibule. None of the animals of this group showed changes in the membranous structures of the vestibule, cochlea, semicircular canals, spiral ganglia or auditory nerve. No evidence of inflammation was seen in the inner or middle ears in this group.

Group III.

Five ears (24 per cent) of the series varying from 48 hours to two months postoperative showed minor to major inflammatory changes (see Fig. 5-b). It is of interest that one animal in the series had both ears operated at different times and on each side a labyrinthitis occurred. Although a suppurative otitis media was present in each animal of this group, all had intact tympanic membranes and no aural discharge was present.

The ears of this group invariably showed dense fibrous tissue invasion through the bony atticotomy defect into the middle ear as in Group II. The tympanic membrane was intact on examination, but the middle ear was filled with a mucopurulent exudate containing numerous polymorphonuclear leukocytes. The middle ear mucosa was markedly thickened, showed vascular engorgement and contained numerous aggregates of lymphocytes. The fibrous membrane over the oval window was no longer a barrier, but instead the tissue encroached upon the vestibule and produced a vestibular fibrosis. In more advanced states this fibrosis filled the cis-



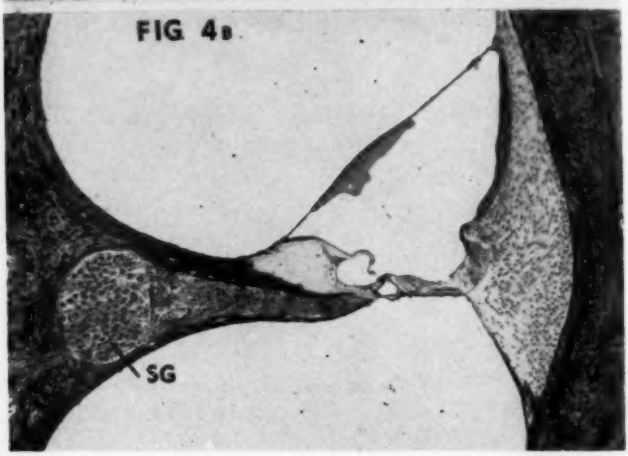


Fig. 4-a. The organ of Corti in the second turn of the cochlea of a cat for which a stapes replacement procedure had been done seven weeks previously. There were no changes in either the vestibular or cochlear end organs. (125X.)

Fig. 4-b. The organ of Corti is well preserved in the basal turn as are the spiral ganglion cells (SG) two months postoperatively. The remainder of the cochlea and vestibular end organs were without changes. In this instance the limbus spiralis shows degenerative changes in the connective tissue cells. This is not an unusual finding as it is known to occur with many different toxic and traumatic agents. Its significance is not completely understood. (68X.)

terna perilymphatica, the ampullae and the semicircular canals. Inflammatory exudate was present in the cochlea to a greater or lesser degree in every instance. Fibrosis did not extend into the cochlea in this series. One animal, sacrificed on the twenty-third postoperative day, showed extensive labyrinthitis and temporal bone destruction. All of the membranous labyrinth and cochlear structures were completely destroyed. Even the modiolus was partially destroyed by an osteitis. Changes varied in the spiral ganglion and auditory nerve in this group. In some instances these structures still appeared microscopically unaltered, but in most partial to extensive degenerative changes were present. The changes in the spiral ganglion were characterized by disappearance of the uniform distribution of the ganglion cells, a reduction in the number of ganglion cells, pycnosis of the nuclei, and a disparity in size of the ganglion cells; the auditory nerve also showed axonal degeneration with residual neurolemmal cells when an extensive labyrinthitis was present.

One animal (4 per cent) did not seem to fall into any of the above categories. It was sacrificed four months postoperatively and grossly a large fibrous polyp was found extending from the middle ear into the bulla. There was a typical suppurative otitis media, but the tympanic membrane was intact, and a well formed membrane was present over the oval window. The vestibular surface of this membrane was lined with intact endothelium. The inner ear was entirely free of any evidence of inflammation or fibrosis (see Fig. 6). There was, however, extensive damage and destruction to the membranous labyrinth. The receptor cells of the macula sacculi, macula utriculi and crista ampullaris were completely gone (see Fig. 7-b). The basilar membrane was intact, but the hair cells and supporting cells had disappeared throughout the entire length of the cochlea, and the spiral ganglion cells showed degenerative changes (see Fig. 7-a).

DISCUSSION.

It is difficult to determine from results of similar procedures described in the literature, the extent or incidence of

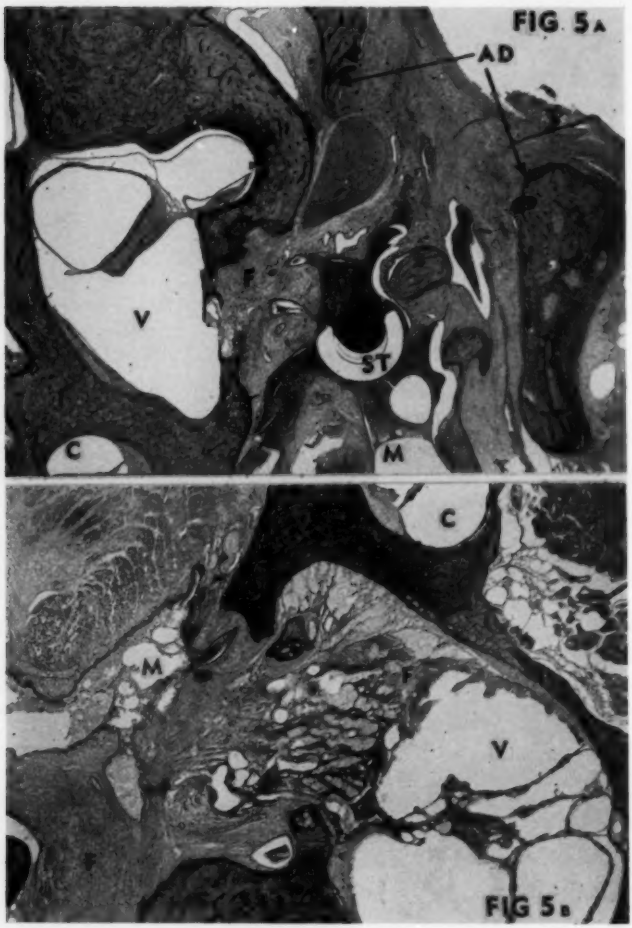


Fig. 5-a. This animal was two months postoperative and is typical of the Group II changes. The middle ear (M) is filled with an aseptic fibrosis (F) but there are no changes or extension of the fibrous tissue reaction into the vestibule (V) or cochlea (C). The footplate of the stapes is fractured with one margin depressed. Fibrous tissue is seen to extend directly through the atticotomy defect (AD) into the middle ear. (15X.)

Fig. 5-b. This cat was 23 days postoperative and is typical of the Group III changes. There is a dense fibrosis (F) in the middle ear (M) with labyrinthitis and extension through the oval window area. (24X.)

fibrosis in the middle ear. Attention has chiefly been directed toward inner ear changes, and only an occasional reference to alterations in the middle ear is found. These are usually concerned with the presence of inflammatory reactions.

In this study an aseptic fibrosis occurred in 27 per cent of the operated animals and in the additional 24 per cent that showed inflammatory changes. In the latter group the middle

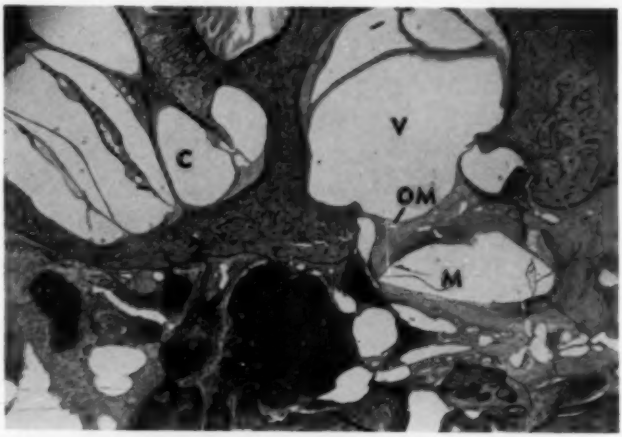


Fig. 6. The middle ear (M) of this animal shows suppurative otitis media, but the connective tissue of the newly formed oval window membrane (OM) has acted as a barrier to the infection. The cochlea (C) and vestibular labyrinths (V) reveal marked degenerative changes in the neurosensory elements four months postoperatively. (15X.)

ear was usually filled with a mucopurulent exudate, and fibrosis was not a predominant feature of the picture. The aseptic fibrosis occurring in the Group II animals can readily be explained on the basis of invasion of fibrous tissue through the bony operative defect. This repeatedly could be demonstrated microscopically. The use of gelfoam over the atticotomy opening did not reduce the incidence of fibrous invasion into the middle ear. It is reasonable to assume that this fibrosis did not necessarily occur in response to the gelfoam or polyethylene, since 45 per cent of the operated ears showed

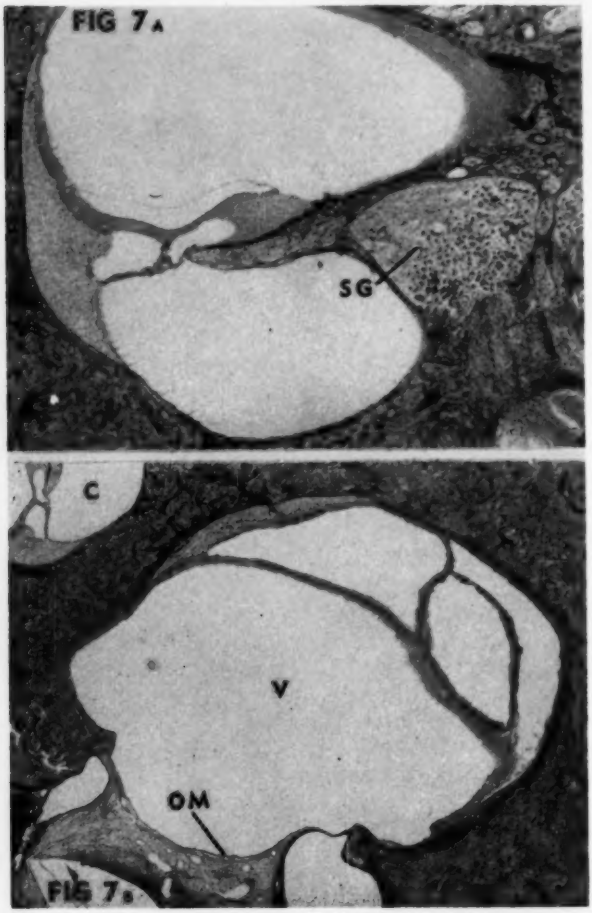


Fig. 7-a. A higher power view of the degenerative changes in the spiral ganglion cells (SG) and organ of Corti in the upper part of the basal turn of the same cochlea shown in Fig. 6. (63X.)

Fig. 7-b. Same as Fig. 7-a, but showing the degenerative changes in the sensory and supporting cells of the maculi of the saccule and utricle. (63X.)

no fibrous change in the middle ear except for occasional delicate mucoareolar strands. These were similar to the fine webs encountered in human middle ears when they are opened for revision of previous stapes surgery.

The changes in the oval window area subsequent to stapedectomy were described by Bellucci and Wolff[3] as spontaneous closure of the oval window with a membrane, in many of their cats. A few developed persistent defects with perilymphatic leakage. In a later study[4] they reported gelfoam as plugging the oval window and breaking off into the cisterna perilymphatica without any evidence of inflammatory or fibrotic reactions.

In the present series the interstices of the gelfoam began to show early invasion of fibroblasts by 72 hours. This progressed rapidly thereafter, and eventually the gelfoam was replaced by a membrane of fibrous tissue over the oval window (see Fig. 3). The middle ear side became covered with mucosa and on the vestibular side was found a membrane of endothelial-like cells continuous with the lining of the vestibule. When an aseptic fibrosis filled the oval window niche on the middle ear side there was still a discrete membrane present on the vestibular side. This membrane seemed to form an impenetrable barrier against fibrous invasion of the vestibule in the absence of infection. In the group III ear in which inflammatory disease was present this endothelial barrier apparently was insufficient to resist invasion of the inner ear by fibrosis.

As early replacement of the gelfoam occurred by fibroblastic invasion a different form of cell was seen to be present along the vestibular surface of the gelfoam insert. Histologically this cell had very sparse cytoplasm and was chiefly characterized by a small spherical basophilic staining nucleus. This type of cell could also be seen in some of the superficial interstices of the gelfoam. These are probably precursors of the endothelial membrane that coalesce on disappearance of the gelfoam.

The gelfoam seems to disappear by lysis. Bellucci and

Wolff[4] demonstrated remnants of gelfoam still present over the oval window as late as two and one-half months after surgery. In this study fragments of gelfoam were in evidence up to approximately two months, following which it underwent complete replacement with a loose areolar tissue (see Fig. 1). No removal of gelfoam by phagocytosis was observed.

The presence of the polyethylene in the middle ear did not seem to elicit any marked reaction about it, except for the formation of a loose fibrous sheath which generally enveloped the tubing. In one animal at four months numerous multinucleated giant cells were found in the lumen of the polyethylene tube. A good opportunity to study the response to polyethylene was afforded in an animal seven weeks after surgery. The strut was found to be free in the middle ear. It was adherent to the wall of the middle ear and surrounded by a very fine covering of loose areolar tissue. Histologically there was no marked reaction or inflammation about it.

According to Schuknecht, et al.,[5] who utilized the vein graft technique, there was no evidence of vestibular fibrosis when the position of the graft was maintained over the oval window, but dislocation of the graft resulted in varying degrees of inner ear damage and fibrosis. Bellucci and Wolff[4] demonstrated fibrosis and extensive new bone formation in the vestibule and cochlea secondary to a labyrinthitis, six months postoperatively. Similar bone formation was not seen four months after surgery in the present series. As previously stated, fibrosis in the inner ear was found only in the presence of inflammatory disease and at the same time there were varying degrees of destruction to the membranous labyrinth apparent. The only instance of destruction to the crista ampullaris, cochlea, saccule, and utricle in the absence of labyrinthitis occurred in association with repeated manipulations at the oval window when an attempt was made to apply a gelfilm disc instead of a gelfoam pad (see Fig. 6). It was technically difficult to keep the strut in place upon the smooth surface of the gelfilm, and it required repeated manipulations. These attempts were finally abandoned, and the gelfoam was substituted. An otitis media with an intact tympanic mem-

brane was present in this animal four months after surgery. No inflammatory reaction was found in the inner ear, and an intact membrane was present over the oval window (see Fig. 7-a).

It is clinically of importance to know the fate of bone fragments which sometimes may fall into the vestibule at surgery. In 1958 Bellucci and Wolff[3] stated that bone fragments dis-

Fig. 8. Bone fragments in the vestibule (V) of a cat three weeks postoperative. Loose strands of connective tissue (A) hold the mass together and connect it with the vestibular wall and the membranous wall of the saccule (S). There is some erosion of the bony fragments and multinucleated giant cells are present. (125X.)

placed into the labyrinth showed evidence of growth. The same authors[4] later described the presence of a stapes head in the vestibule without any untoward reaction. In one specimen of the present series a fragment of the footplate was found to have lodged in the vestibule. It was adherent to the wall of the vestibule in the vicinity of the saccule. There was a minimal fibrosis about the fragment sufficient to make it adherent to the vestibular wall, but no reactions in the cisterna perilymphatica were present (see Fig. 8).

Bellucci and Wolff[4] frequently referred to a fibrosis of the

cochlea and cited instances of extensive cochlear damage due to labyrinthitis. They described degeneration of the organ of Corti in the basal turn as a result of stapedectomy and suggested that the lytic action of gelfoam in the vestibule and cochlea might be responsible for further destruction of the organ of Corti. Schuknecht, et al.,[8] described damage to the organ of Corti, secondary degeneration of spiral ganglion cells and rupture and collapse of Reissner's membrane in the basal turn, after fracture-dislocation of the stapes footplate in cats. They also found otitis media and labyrinthitis without cochlear fibrosis three months after surgery, and with the presence of a permanent oval window fistula. The labyrinths and cochleas in the groups I and II animals of this series showed no change by routine histologic methods. It was concluded that trauma due to manipulation was not present in these groups. Extensive damage to both the membranous labyrinth and the cochlea was found in the group III animals in which labyrinthitis was present. These changes varied from loss of the neurosensory elements in the saccule, utricle, crista ampullaris and organ of Corti to complete degeneration of these organs. The vestibular and ampullary structures often underwent fibrous replacement. The absence of fibrosis in the cochlea in group III animals was unique. It was present in the vestibule, ampulla and semicircular canals with inflammatory disease, but peculiarly no fibrosis was found in the cochlea as long as four months after surgery. This does not imply that it might not occur eventually.

Rupture of Reissner's membrane was not present in our series unless the membranous structures had been damaged by an inflammatory or traumatic agent. It was intact in the group I and II animals.

Schuknecht, et al.,[8] described ganglion cell degeneration secondary to labyrinthitis three months postoperatively. Bellucci and Wolff[4] found reduction in the size and bizarre shapes in the ganglion cells, vacuolization of the cytoplasm and enlargement of the satellite cells and auditory nerve changes. In the present series spiral ganglion and auditory nerve changes were found only in association with advanced labyrinthitis and in the one instance of trauma.

With depression of the stapes footplate Schuknecht, et al.,[5] described the formation of a muco-endosteal membrane between the footplate and the oval window margin. They reported damage to the organ of Corti in the upper basal turn due to excessive footplate manipulations and recommended that this procedure be discontinued. Bellucci and Wolff[3] described distension of the scala media with displacement of Reissner's membrane, damage to the organ of Corti and alteration of the limbus following procedures that depressed the footplate. Several footplates of this series were found to be depressed into the vestibule; but no damage was observed either to the vestibular or cochlear labyrinths, and a fibrous endothelial membrane bridged the gap between the depressed footplate and the window margin (see Fig. 2-a).

Two months following stapedectomy, degenerative changes were seen in the connective tissue cells of the limbus spiralis (see Fig. 4-b). Since this is not an unusual finding seen in association with many different toxic and traumatic agents, it was difficult to evaluate its significance.

The term muco-endosteal would infer that the membrane which frequently spans a defect in the oval window derives its origin from the endosteum. It seems that this membrane is a continuation of the endothelial lining of the vestibule; consequently, reference is made in this report to this structure as an endothelial-like or muco-endothelial membrane.

SUMMARY.

1. Stapedectomy and stapes replacement can be carried out in cats, using the gelfoam and polyethylene strut technique without demonstrable microscopic changes in the inner and middle ear postoperatively. Apparently the presence of the foreign materials *per se* does not stimulate fibrosis.

2. Fibrosis in the middle ear in cats appears to be the result of invasion of fibrous tissue through the atticotomy defect and can occur in the absence of sepsis or inflammation. There is no apparent correlation between fibrosis in the middle ear and inner ear damage.

3. The membrane formed over the oval window after a stapes replacement procedure is apparently a barrier to fibrous tissue invasion of the inner ear; however, it does not prevent the invasion of an infection into the labyrinth.

4. Inner ear changes are found to occur in the presence of postoperative sepsis and undue surgical trauma over the oval window area. Trauma over the oval window area is capable of producing degeneration of the receptor cells in the saccule and utricle, as well as destruction of the organ of Corti, and spiral ganglion degeneration.

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GILL MEMORIAL EYE, EAR AND THROAT HOSPITAL.

The Gill Memorial Eye, Ear and Throat Hospital will hold its Thirty-Fourth Annual Spring Congress in Ophthalmology and Otolaryngology and Allied Specialties, April 10 through April 15, 1961. There will be 20 guest speakers and 50 lectures. For further information contact Dr. E. G. Gill, 711 So. Jefferson St., Roanoke, Va.

ABERRANT SALIVARY TUMORS: A REVIEW WITH PRESENTATION OF SIX CASES.*

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Tumors of salivary gland type, outside the major salivary glands, are relatively rare and usually of low-grade malignancy. There has been a tendency toward conservatism in their treatment. More radical parotid surgery is credited with lowering the recurrence rate, previously stated as 25-40 per cent,[1,2] to less than 5 per cent.[3] It has been stated that the aberrant salivary tumors are more malignant than those in the major glands,[4] yet many recent papers continue to describe simple enucleation as the treatment of choice. It is the purpose of this paper to review the common salivary tumors with emphasis on the aberrant locations and to determine what constitutes adequate treatment in these locations.

Six patients will be presented, two with nasal cavity tumors, two with palatal lesions, one in the upper alveolus, and one in the tongue.

CLASSIFICATION.

Histologic classification of salivary tumors has been complicated by conflicting terminology, and the difficulty of correlating the microscopic picture with the clinical course. The word "benign" when used in reference to mixed or mucoepidermoid tumors means "relatively favorable," as all of these tumors are known to recur, to invade, to metastasize, and to kill. Table I shows some of the proposed schemes of classifications. Morphologic distinction is difficult in this pleomorphic continuum, and the phrase "mixed tumor" is often used to describe any member of the group, as well as a specific type. A 15-year-old term "mucoepidermoid tumor"

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and the 100-year-old term "cylindroma" (adenoid cystic carcinoma) are simply not used by some pathologists.

THE MALIGNANCY OF THE SALIVARY TUMORS.

These tumors have been called the most benign malignant group, or the most malignant benign group. Their natural history is often measured in decades, and recurrences have been reported after 40 years. Histologic criteria of prognostic

TABLE I.

Classification of Common Salivary Tumors.

This Paper	Lund[3]	Vellios[6]	Willis[7]
Benign Mixed Tumor	Adenocarcinoma—*in situ*	Benign	Pleomorphic Adenomas—Adenocarcinomas
Malignant Mixed Tumor	Adenocarcinoma—*in situ*	Malignant with Slow Course	Pleomorphic Adenomas—Adenocarcinomas
Cylindroma	Adenocarcinoma—*in situ*	Malignant with Slow Course	Pleomorphic Adenomas—Adenocarcinomas
Mucoepidermoid	Secreting Epidermoid Carcinoma	Malignant with Slow Course	Pleomorphic Adenomas—Adenocarcinomas
Adenocarcinoma	Adenocarcinoma	Malignant with Rapid Course	Pleomorphic Adenomas—Adenocarcinomas

value have been said not to exist. McFarland[1,2] suggested as bad prognostic signs, a preoperative duration measured in months, rapid growth, lymph node metastases, and histologic areas of frank cancer. These patients often die within the year.

The mixed tumor is especially difficult to prognosticate, and the phrase has been called a harmful misnomer for a carcinoma.[5] Metastasis is very rare in the most benign-appearing mixed tumors, but has been reported. In 1943, Mulligan[8] collected 21 cases of true metastases in mixed tumors, two of which were ectopic (palate). In the Memorial series[3] there is only one case of metastasis of a benign mixed tumor, but a much higher number in all of the other forms.

A greater relative malignancy of the cylindromas is the

chief cause for distinguishing this form. Metastases occur in 30 per cent,[3] but uncontrolled local disease is the usual cause of death. In their series of ectopic tumors[9] McDonald and Havens found that whereas at five years more patients with cylindromas were alive than with adenocarcinomas; by eight years this situation was reversed; 48 per cent of the patients with adenocarcinoma was alive versus 31 per cent of those with cylindromas. They showed graphically that the death rate of cylindromas continued to drop after five years, in a straight line fashion, rather than leveling off as with most malignancies. This illustrates once again the fallacy of using a "five-year-cure" as a definite point. In many tumors, two-year results are almost as significant as five, but in the salivary tumors, thyroid tumors, and many irradiated tumors, a much longer interval is necessary before one speaks of cure. They felt the perineural invasion was the common cause of the recurrences, and that any pain or paralysis indicative of neural involvement would be a bad prognostic sign.

Russell[10] felt poor encapsulation was the chief cause of cylindroma recurrence, and she noted that whereas 17 of 18 palatal mixed tumors were alive and well over five years, only seven of the 17 palatal cylindromas were alive and six of these inoperably recurrent. As these were all equally accessible on the palate, she did not feel that surgical inaccessibility was very important.

Ranger[11] felt that the fact that the common ectopic sites were hidden, as well as possibly of a more sinister cell type, might account for this increased malignancy. Over 50 per cent of his series of 80 ectopic tumors had already recurred within the one to two year interval he had followed while writing his paper. None of his 11 lip tumors recurred, as the location was easily accessible, and ten of the tumors were the more innocent mixed form; the basalioma (cylindroma) form within the buccal and nasal cavity accounted for most of the recurrences.

Infiltration as the cause of cylindroma recurrences had been stressed by Putney and McStravog,[12] and Berdal and Mylius[13];

the latter have stated that there is a "tendency to infiltration, which by far exceeds what we previously had imagined."

INCIDENCE.

It is difficult to get an accurate idea of the over-all incidence of these tumors in the general population. The longer reported series tend to come from Tumor Centers where there has been a high degree of preselection, and the shorter series lack statistical significance.

Owens[14] found no cases of aberrant salivary tumors in over 58,000 cases processed by the Surgical Pathology Department at Los Angeles County Hospital in 1948. The Radiumhemmet treated 756 consecutive salivary tumors, from 1910 to 1940, of which 22 per cent were ectopic.[15] Of 5,000 anterior fore-gut specimens examined at Christie Cancer Hospital, from 1944 to 1953, 272 salivary tumors were examined, in which 82 were ectopic.[10] Twenty-two of the 62 salivary tumors treated at the Tumor Clinic of the Episcopal Eye, Ear and Throat Hospital were ectopic, probably a higher incidence than seen in general hospitals.

SEX AND AGE.

There is almost universal agreement in long series that females predominate in a range of 55 per cent, not far different from the general population of the "cancer age."

There is similar agreement that the predominant age-group is the fourth and fifth decades; patients having had the tumor for several years when first seen. Foote and Frazell[3] state that their malignant mixed tumors occurred in patients ten years older than their benign cases; this supports their view that malignant tumors arise from previously benign tumors.

RACE.

Most reports concern primarily European groups. Schulenberg[16] studied 84 cases of mixed salivary tumor in Pretoria and found the incidence in the Cape Town natives (not racially

mixed) five times as high as in the European. Only 46 per cent of these tumors were in the parotid, with 19 per cent submaxillary, 17 per cent palate, and 18 per cent other ectopic sites.

Marsden,[17] studying salivary gland tumors in Malaya, found their incidence 4½ per cent of all tumors seen, with 63 per cent in the parotid, 30 per cent in the submaxillary gland (highest in any series), 3½ per cent in the palate, and other ectopic sites 3½ per cent.

Sharma[18] saw 50 cases in Nagpur, India, and found the average age to be only 23 years, with 12 per cent on the lips.

Hickey[19] noted that in the pathological laboratory in Khartoum, there were 37 salivary tumors in the same interval as 69 breast carcinomas, the most common malignancy of that area.

CASE REPORTS.

Case 1. A 56-year-old white housewife developed increasing left nasal obstruction, tearing, and recurrent epistaxis from January to May, 1956. Another specialist found a large, granular, ulcerated polyp, arising in the left inferior meatus, extending up to the middle turbinate. A polypectomy removed "8 irregular, firm, mottled, pinkish-red fragments, measuring from 0.5-2.5 cm., with small secreting areas 0.1-0.2 cm. in size." The histologic report on all sections was "malignant mixed tumor." Sinus films were normal. On examination in August, 1956, a grey plaque was seen under the inferior turbinate, and I performed a left lateral rhinotomy removing *en bloc* the mucosa of the nasal floor, the inferior turbinate, the preturbinal region, the nasolacrimal duct, the nasoantral wall, and the inferior portion of the ethmoids. Despite the loss of the left bony pyriform aperture, there has been no deformity. The patient was asymptomatic, and without evidence of disease when seen June 25, 1959.

Comment. The brief period of symptoms, and the ulceration, are compatible with the "malignant" designation assigned by the pathologist. The wide margin of the resection and the lack of disease in the antral mucosa suggest a favorable prognosis. As tearing was her initial symptom, and also the initial symptom two months after her polypectomy, I feel her tumor arose in the nasolacrimal duct.

Case 2. A 48-year-old white housewife developed pain below the right eye, and increasing right nasal obstruction in August, 1956. In October, her sinus X-rays showed diffuse clouding of the right frontal, ethmoid, and maxillary sinuses, without fluid levels, or bone destruction. Another specialist performed a right Caldwell-Luc and intranasal ethmoidectomy,

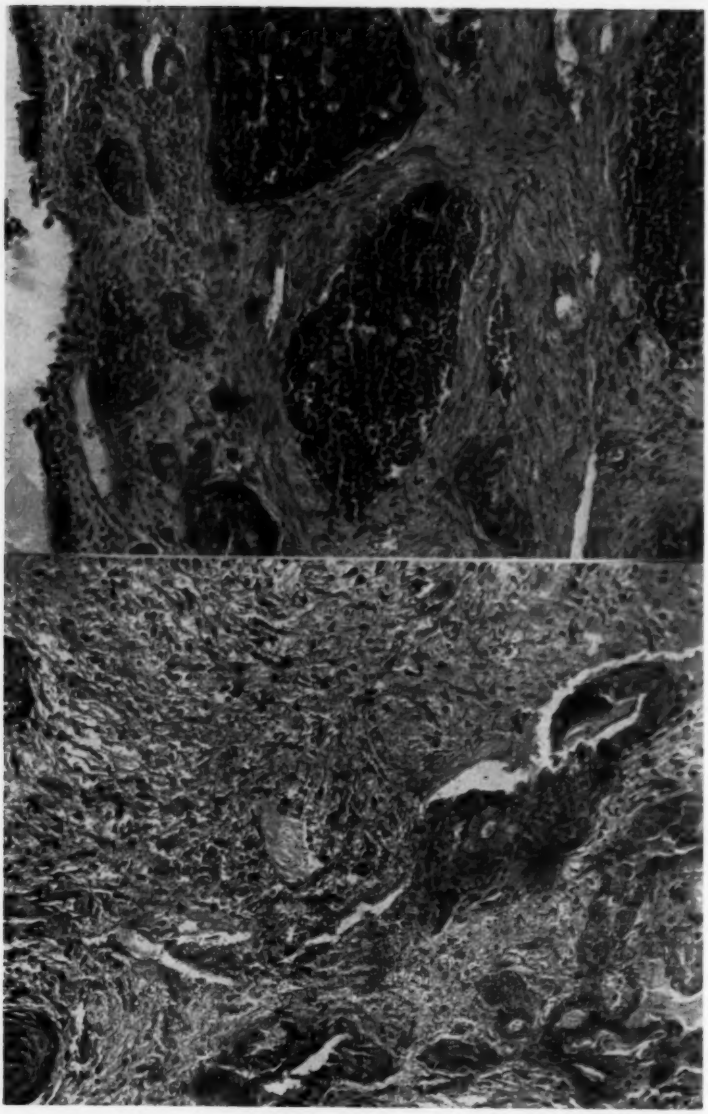

FIG. 1. Case 2. Left: the essentially benign appearance of the middle turbinate biopsy with myxomatous tissue and well differentiated adenomatous cells. Right: nests of adenocarcinoma from the ethmoidal region at rhinotomy.

removing a large polypoid mass in the middle meatus, reported as "mixed tumor, histologically benign." Her pain continued and tearing developed, and a repeated deep biopsy from the middle turbinate on October 26, was reported by a different pathologist to be a benign mixed tumor (see Fig. 1).

I first saw the patient in December, 1956, with a firm red, granular tumor mass, apparently arising from the middle turbinate. Further surgery was agreed upon, but permission for possible eye removal was not obtained. A lateral rhinotomy was performed, and gross tumor was seen to extend into the cribriform plate as well as the orbit posteriorly, and superiorly into the frontal sinus. A frozen section revealed adenocarcinoma, grade 2. She was considered unresectable, and at her request, was transferred to the National Cancer Institute, where a neurosurgical exploration was performed on January 9, 1957. The dura mater was found to be involved by poorly differentiated adenocarcinoma. She received radiotherapy and nitrogen mustard with little response, and expired on May 8, 1957, with pulmonary and cervical metastases, as well as uncontrolled local disease.

Comment. None of the films prior to the December operation were suggestive of malignancy, nor were the biopsies. Tomograms or stereo views might have shown some of the destruction present. The benign histologic appearance of biopsies taken from the antral mucosa, and middle turbinate gave poor indication of the undifferentiated adenocarcinoma found in the deeper portion of the tumor. The dural extension seen three weeks later confirmed the lack of resectability in this patient. Lack of permission to remove the eye was a handicap, limiting surgery to an intermediate, rather than radical approach, when a malignancy was proved.

Case 3. A 69-year-old white male was seen in April, 1957, for a painful ulcerated mass in the left tongue, of three months' duration. The discrete mass was ulcerated along its lateral edge, with the surrounding epithelium leukoplakic (see Fig. 2). Two punch biopsies showed mixed tumor. The Wassermann was positive. I performed a sub-total glossectomy and upper neck dissection; the final diagnosis was "adenocarcinoma of a salivary type" (see Fig. 3). The postoperative course was uneventful with fair speech and good swallowing. There was no evidence of disease when seen in April, 1959.

Comment. Clinically, this patient was a moderate pipe smoker, with a positive serology and a leukoplakic ulcerated mass, all suggestive of epidermoid carcinoma. A frozen section at the time of surgery confirmed the true salivary nature of the tumor, so that only an upper neck dissection for exposure was performed, rather than a total radical neck dissection as would be done for an epidermoid carcinoma. As in Case 2, there was a great difference between the biopsy and

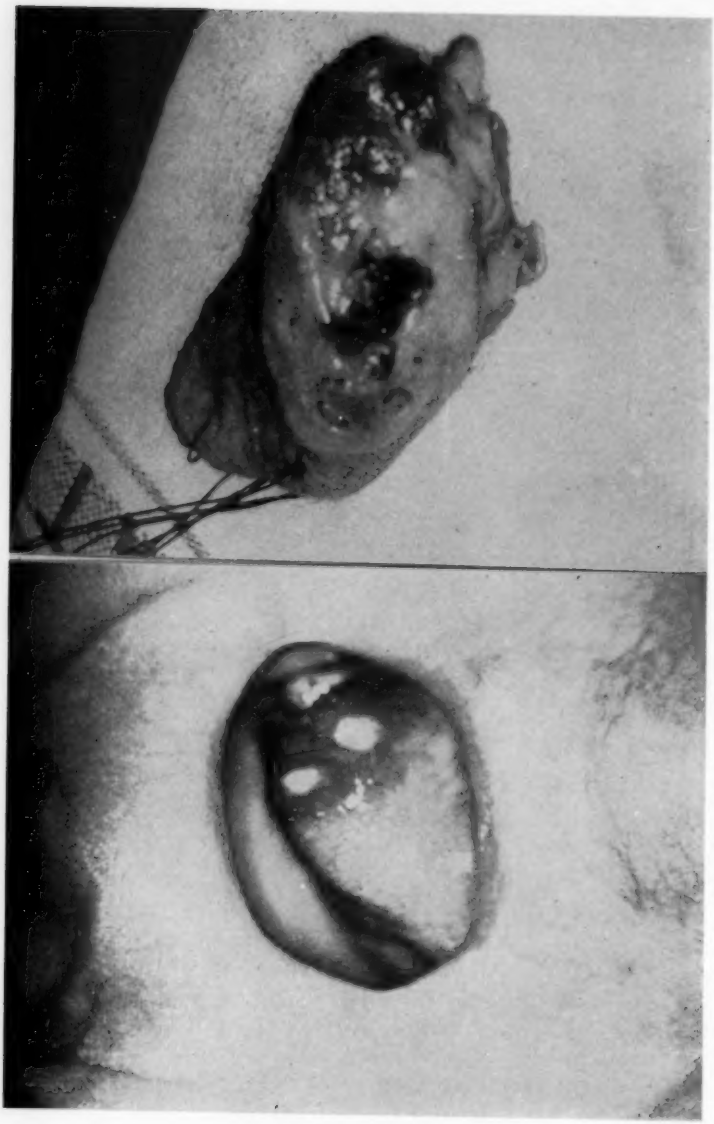

Fig. 2. Case 3. The ulceration and leukoplakia gave the clinical impression of an epidermoid carcinoma.

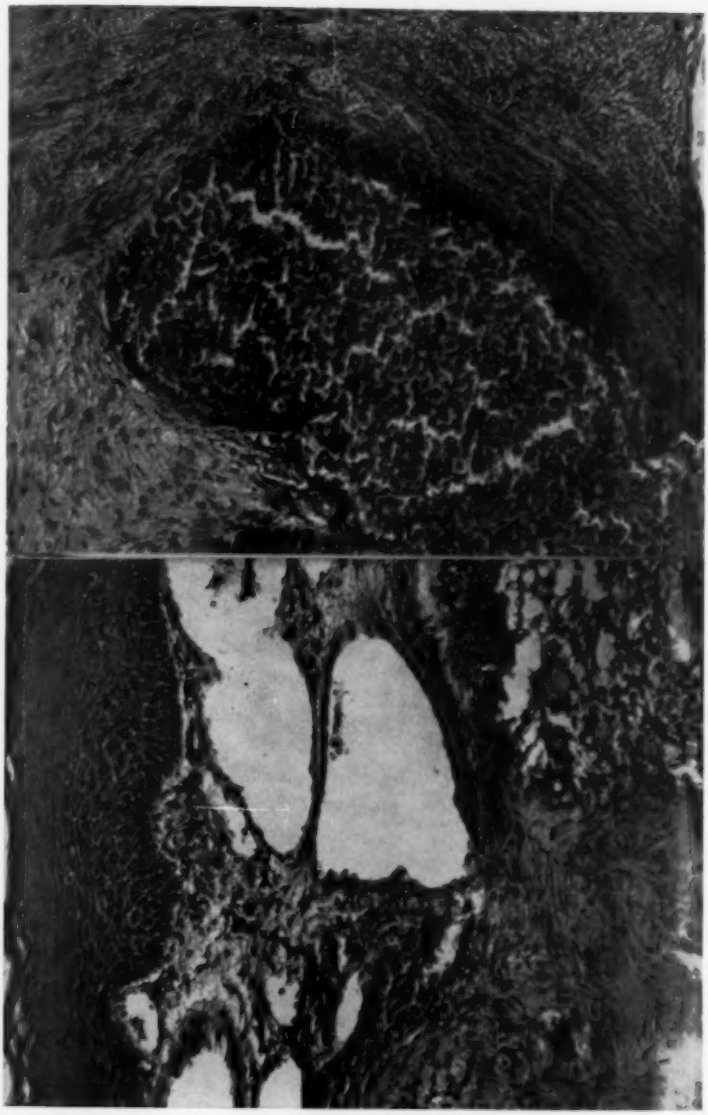

Fig. 3. Case 3. Left: biopsy from tongue reported as mixed tumor. Right: main tongue specimen reported as adenocarcinoma.

the final tissue diagnosis. These cases illustrate the continuum of morphology that may occur with these lesions, and the lack of reliability from a biopsy diagnosis alone.

Case 4. A 68-year-old white male complained of a "fluffy" feeling in the left palate, and discovered a smooth mass in the left upper alveolus, in September, 1957. A surgeon biopsied and cauterized this mass, reported as "mixed tumor." Sinus X-rays were normal. When I saw the patient, there was a deep ulceration in the left upper alveolus, with an obvious burn extending into the buccal gutter. On October 1, 1957, I performed a left partial maxillectomy with the cheek mucosa in continuity through a Weber-Fergusson incision. The remaining antral mucosa appeared normal, but was curetted. Sections showed "mixed tumor of low-grade malignancy," with marked extension into the perineural lymphatics, and widespread replacement of the marrow spaces, and extension into the periosteum and supporting stroma of the antral mucosa (see Fig. 4). An obturator was fitted, and their had been no evidence of recurrent disease, when last seen on June 24, 1959.

Comment. Although called "mixed tumor" by the pathologist, many areas have a typical cylindromatous pattern, which is in keeping with the perineural invasion. The earliest symptom of a "fluffy" paresthesia of the palate may have arisen from this perineural invasion, and sensory and motor neural changes are looked for to presage any recurrence. A preoperative radiological evaluation gave no warning of the antral mucosal invasion, nor did the antral mucosa appear unusual at operation, as the tumor had infiltrated submucosally, by direct extension through the marrow spaces. Although free of disease for two years, the notorious late recurrence of cylindromatous lesions, and the many foci of perineural extension, makes one hesitant regarding his prognosis.

Case 5. A 30-year-old white housewife had a mass curretted from her right hard palate in May, 1955, by her oral surgeon. This recurred as a non-ulcerated, painless, multilobular mass, which was excised locally and reported as "mixed tumor," in May, 1958. I saw her following the second operation, with a large operative defect tending to heal (see Fig. 5). Sinus X-rays showed a "polypoid soft tissue density in the inferior portion of the right antrum." Assuming this to be a direct extension of the tumor, I performed a classical right radical maxillectomy, preserving the orbit. The polypoid thickness proved to be inflammatory and no further tumor was found in the specimen. The patient has adjusted well to her obturator and was free of disease when last seen on July 22, 1959.

Comment. This patient had a more radical removal than Case 4, on the basis of her positive antral X-ray findings and my experience with the great extension in the previous case. A comment applicable to Cases 1, 2, 4, and 5, is that in each

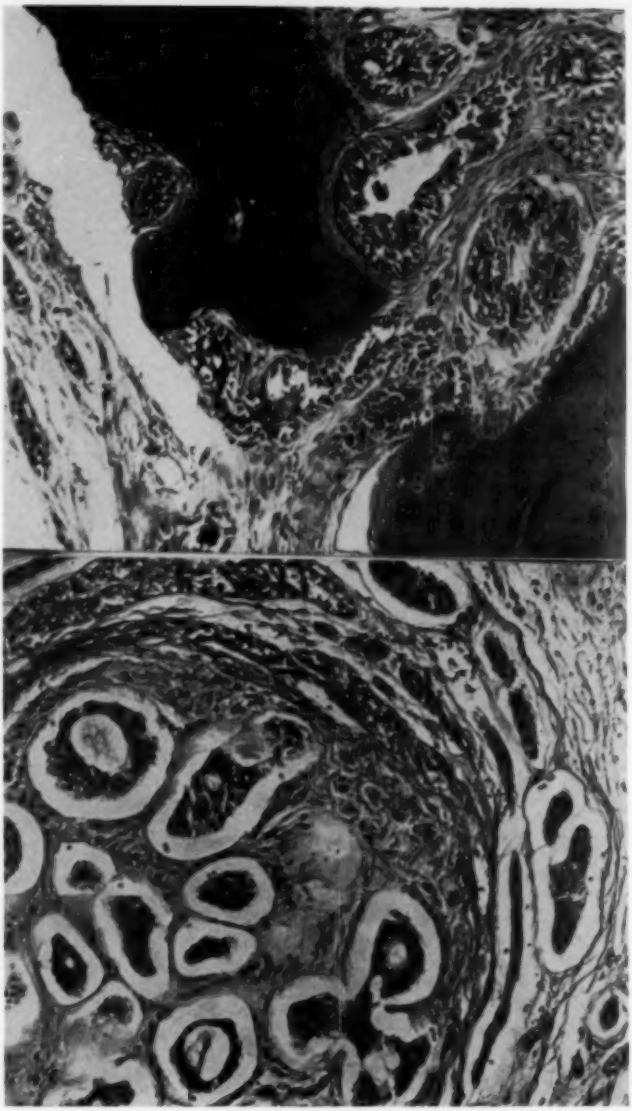

FIG. 4. Case 4. Left: perineural invasion, frequently seen in cylindromas. Right: bone marrow invasion; tumor cells extended from the alveolus into the antral mucosa.

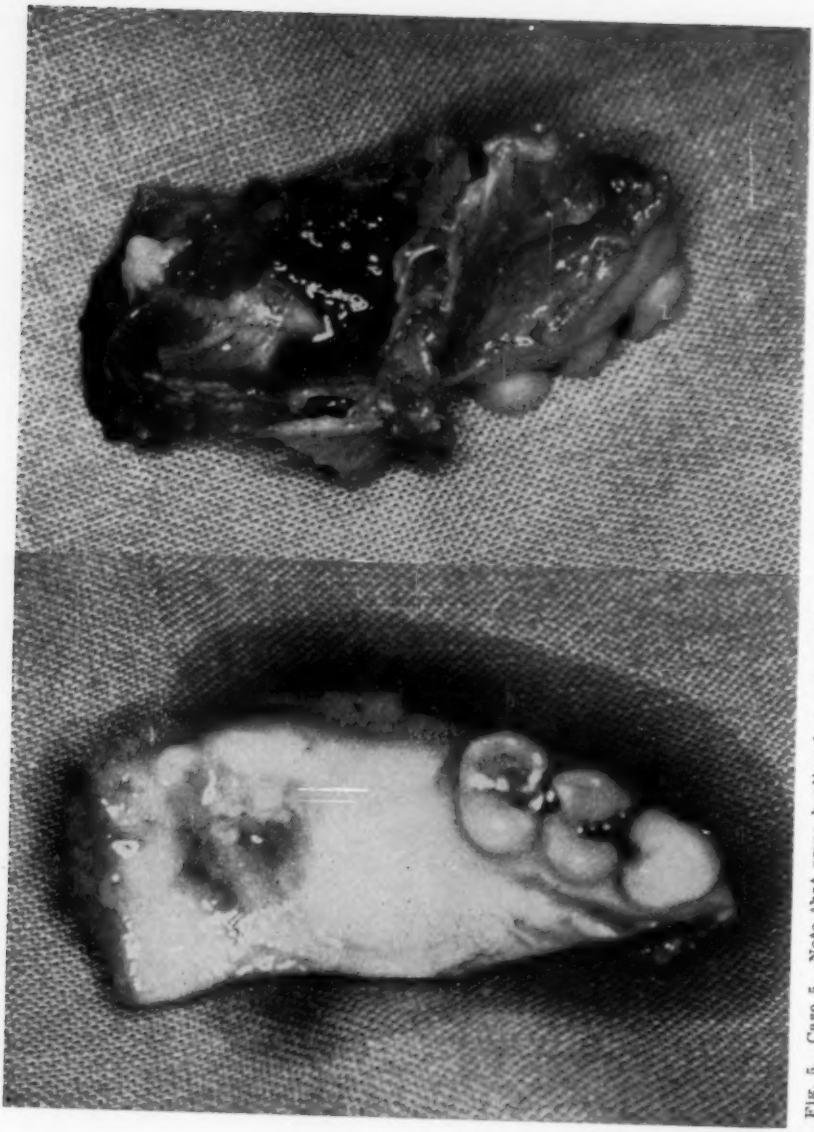

FIG. 5. Case 5. Note that some healing has occurred in the biopsy site. The thickened antral mucosa, considered tumor extension from the X-ray appearance, proved to be inflammatory.

instance, the appearance of the lesion had been markedly changed by excisional biopsies prior to my seeing the patient.

Case 6. A 45-year-old white housewife was referred by her dentist in October, 1958, for evaluation of a firm, smooth mass in her palate, from the midline to the alveolus, allegedly present 20 years. X-rays were normal. Two punch biopsies were taken, reported as mixed tumor. She has refused any further surgery unless her dentures become ill-fitting, indicating growth. There had been no change when seen in May, 1959.

Comment. I feel that this patient does have a mixed tumor, which has lain dormant for many years. No growth potential is evident at present. Although the tumor seems well separable from the palate medially, laterally it feels fixed. This loss of definition laterally would necessitate some removal of palatal bone to effect a cure. A 20-year history of inactivity has caused the patient to reject surgery, either the enucleation preferred by the referring oral surgeon, or the resection

TABLE II.

The Increased Malignancy of Ectopic Sites.

	Benign and ? Benign	Semi-malignant, Potentially Malignant	Known Malignant
Broadbent[22]	15	10	8
Owens[14]	250	122	652
Ranger[11]	41	33	6

I suggested. It is felt that something as sensitive as the fit of a denture should give early warning of growth.

DISCUSSION AND CONCLUSIONS.

This review was instigated by experiences with six private patients, referred for definitive tumor therapy for aberrant salivary lesions. Four proved to have histologically malignant tumors, one benign, but recurrent within three years of initial removal, and one quite benign.

There is an increased incidence of histologic malignancy in salivary tumors in the ectopic sites; demonstrated in these six patients, in Table II, and in the body of this paper. Possible explanations for this increase in malignancy are: *1.* predominantly mucoid glands of the ectopic sites may generate

Fig. 5. Case 5. Note that some healing has occurred in the biopsy site. The thickened antral mucosa, considered tumor extension from the X-ray appearance, proved to be inflammatory.

more malignant tumors than the serous parotid, or mixed submaxillary glands; *2.* whereas the parotid and submaxillary cells are rarely exposed to anything but their own secretions, the superficial locations and short duct system of the minor glands of the upper air and food passages expose them to a variety of exogenous irritants; *3.* without postulating any ontogenetic embryonic cell rests, it may be said that the minor glands are phylogenetically older and simpler, and hence, probably more capable of de-differentiating into neoplasia than their well differentiated major gland homologs.

In the same way that a well differentiated hyperkeratotic papilloma of the larynx may gradually, or abruptly, become carcinomatous, so may salivary adenomatous tissue give rise to adenocarcinoma. In each case, any degree of malignancy is possible. It is the peculiarity of mature salivary glandular epithelium to form its own specialized secretion. When not connected with a patient's duct system, the secretion necessarily will be lessened in quantity and of abnormal quality. Its lability has been shown by variable staining techniques; presumably a chondromucin is formed, which may be subject to organizer influence and form pseudo-cartilage, but rarely bone.[20,21] This secretion separates the cells and cell aggregates haphazardly, thus giving the pleomorphic appearance known as mixed tumor. The less mature tumor cells are less capable of this secretion and may group as sheets of nonfunctioning adenocarcinoma. All variations from malignant mixed tumor to pure adenocarcinoma are seen often in the same specimen. The highly malignant tumors resemble anaplastic carcinoma or sarcoma and may give no clue as to their origin in salivary tissue.

Mucoepidermoid tumors properly are a distinct type, arising from the salivary ducts; cell proliferation is seen in these ducts, and the intermediate cells, characteristic of the tumor, have been found in this location.

A cylindroma is more mysterious; pseudo-cylindromatous patterns are not uncommon in mixed tumors, but the pure cylindroma has more regularity of pattern. The secretion of its strands of cells is apparently limited to the regular

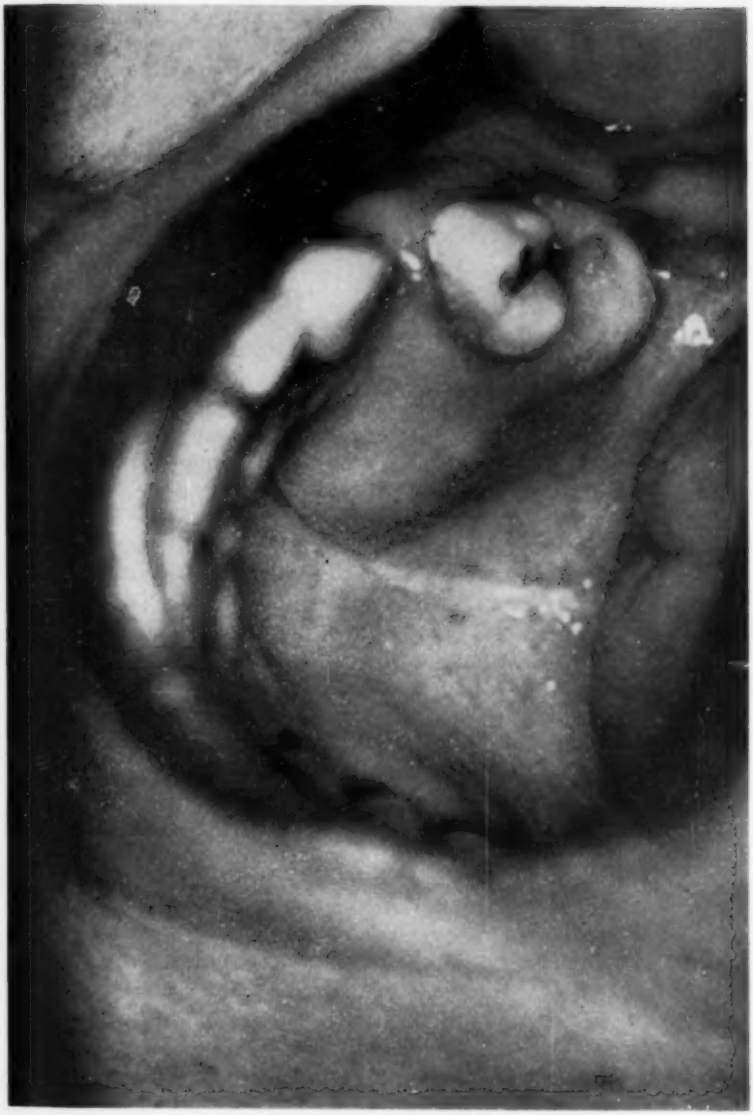

Fig. 6. Case 6. This patient claims there has been very little change in this palatal tumor in 20 years.

hyalin stroma, which does not further differentiate. Its great malignancy and tendency for perineural invasions have been frequently commented on but never well explained. Its scanty secretion might imply a ductular rather than acinar origin. Although occurring in the serous parotid, it is most common in the mucous glands. It has not had the histochemical and tissue transplant investigation the mixed tumors have had. The loose definition of mixed tumor, as comprising, *1.* epithelial, and *2.* connective tissue elements should be abandoned; the mucoepidermoid tumor and cylindroma should be distinguished when possible by pathologists and clinicians.

A representative biopsy of these submucous tumors should be obtained. Little objection could be made to aspiration biopsy, or use of a fine punch, even when necessary to break the capsule at a superficial place. The most useful information obtained on biopsy could be that one is indeed dealing with a salivary tumor; since the degree of malignancy is not predictable, such words as benign or malignant lose their meaning. If the capsule is entered for biopsy, a wide capsular resection should be planned, to include the original biopsy site. The removal of the entire visible lesion for biopsy, and then referral to a second physician, who has never seen the patient, is objectionable; there should either be a smaller biopsy, preliminary consultation, or the first surgeon should be prepared to give definitive treatment. Unlike biopsies of ulcerated lesions which may be taken from the area most likely to show the malignancy, submucous biopsies from the most superficial areas can offer little information other than that for histologic malignancy in the small area submitted.

All possible information from roentgenography, including tomograms and stereo views should be obtained on any tumor not obviously limited to the soft tissues. A careful neurological examination is of benefit in cylindromas. It is my feeling that in general, the increased malignancy arises not only from the cell type itself but also the failure to do a wide enough resection. The surgery for any aberrant salivary tumor, of recent origin, or recent growth, or recurrence, should be as

great as though the primary tumor were an epidermoid carcinoma. I do not feel that neck resection should be performed, however, unless enlarged nodes are present. Death occurs from uncontrolled local disease and not metastases.

It is concluded that aberrant salivary tumors are not mysterious and probably are seen more often by otolaryngologists than any other medical specialists. Making use of nose, lip, and cheek flaps, excellent exposure is obtainable in the anterior locations, allowing easy, wide resection. As in the parotid gland, wide resection should greatly improve the cure rate over enucleation. There will still be some patients whose tumors are so superior, or so posterior, as to render wide resections impractical.

Radiotherapy and chemotherapy are of little use in palliation, for which it is hoped, there will be less need in the future—if more radical surgery is adopted.

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GOULD FOUNDATION AWARD.

The William and Harriet Gould Foundation announces the winner of the Gould Award for 1960: Dr. Tanwillen van den Berg of the University of Groningen, The Netherlands. The Award was presented to Dr. van den Berg in recognition of his contributions to laryngeal physiology.

The Gould Award is presented annually for outstanding contributions to basic laryngeal research. The candidate is selected by an International Committee, which at present consists of Professor Cotoji Satta, M.D., Tokyo, Professor Luzius Rüedi, Zurich, Professeur Georges Portmann, Bordeaux, and Dr. Hans von Leden, Medical Director, 30 North Michigan Avenue, Chicago 2, Ill. The Award includes an illuminated plaque and a cash prize of $350.

A STUDY OF THE EFFECTS OF LOUD SOUND ON TINNITUS.*

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and
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INTRODUCTION.

Subjective tinnitus aurium in many cases appears to be due to irritability or hyper-irritability of a partially damaged cochlea. Theoretically it should be possible to alleviate the condition by destroying or fatiguing the involved nerve endings. Selective destruction of portions of the cochlea by exposure to loud sound has been amply demonstrated in animal experimentation.[2,16,17,21] A review of the literature reveals that when the human ear is exposed to a tone of known frequency and great intensity, the result is a loss in hearing acuity beginning about one-half octave below the traumatizing tone and extending to all frequencies above it, with the greatest threshold shift occurring about one octave above.[2,17] In a preliminary study of 200 patients with tinnitus[15] we noted that the central frequency in 70 per cent of the cases was above 3,000 cycles per second, a finding confirmed by other reports.[5,13,18] It would appear that the majority of patients could be exposed safely to a pure tone, lying at or above the pitch of their tinnitus, in an effort to fatigue the involved portions of the cochlea, without further damage to their hearing in the speech range.

A search of the literature lent enough support to this theory to merit further investigation. Jones and Knudsen in an article written in 1925[12] describe an instrument for "bombardment of the cochlea" to "de-sensitize (it) to certain tonal regions, the regions corresponding to the tinnitus." As a

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result of cochlea bombardment they at times noted a "lessening of the tinnitus," but further state that this "acute phenomenon, which it undoubtedly is, does not last." As this is the only reference in the literature to a purposeful attempt to treat tinnitus with loud noise, it is unfortunate that they did not go into greater detail and did not report the intensities used.

There are, however, reports of incidental observations of the effect of noise on tinnitus. Bouchet[1] in 1952 described a case of a 62-year-old man who was "cured" of tinnitus of six months' duration by sudden, accidental exposure to the loud noise of a buzz saw in a lumber yard. The intensity of the noise was sufficient to render the man unconscious, and upon recovering, he discovered that his tinnitus was completely gone. Spaulding in 1903,[19] reporting on the usefulness of studying tinnitus by matching its tone with those of a piano, made the observation that exposing the ear to the prolonged action of a similar tone on an "organ or reed pipe or violin string" diminished or completely removed the tinnitus for a certain length of time. Timofeyeff, in 1955[20] in a paper discussing his finding that it was necessary to use 70 or 80 decibels or more of sound to mask subjective tinnitus, also noted that in some cases the tinnitus disappeared for one to three minutes after cessation of the "covering sound."

The theory of partial cochlear fatigue could explain the facts in each of these reports and observations. It seemed reasonable to conclude that with modern audiological and psychoacoustical equipment it would be possible to control this phenomenon for therapeutic purposes.

METHODOLOGY.

It is immediately obvious that "acoustic therapy" as we have come to call it, could be beneficial only in those cases in which the tinnitus arises in the cochlea. As Goodhill[10] and Fowler[8,9] have stressed repeatedly, the causes of tinnitus aurium are many and varied; however, we agree with Eigler[4] that the majority of subjective auditory sensations have their source in the end-organ. In our study of 200 patients we

found that about 75 per cent were probably of cochlear origin.[15]

For the present study we attempted to weed out all patients whose tinnitus was not likely to have its etiological source in the cochlea, recognizing, of course, that this is difficult to do with exactness. The classification of a given case of tinnitus as cochlear or non-cochlear in origin was based on many factors: a careful perusal of the patient's history, physical and otologic examination, the nature of the hearing loss, and

Fig. 1.

the frequencies involved in the tinnitus. In general, the source probably lies in the end-organ, if the following criteria are met: the involved ear appears normal on examination; the patient has no history of otologic pathology or of head injuries or other physical trauma associated with the onset of the condition; he has tinnitus in one ear only, or, if in both, he hears a different sound in each ear: he has a perceptive hearing loss in the involved ear with a drop at approximately the region of his tinnitus; the sound he hears is made up of a relatively narrow band of frequencies; and the phenomenon of recruitment can be established reliably.

In addition to the criterion of cochlear origin, only those patients were selected for treatment whose tinnitus was composed of frequencies lying above 3,000 cycles per second or who had more than a 70 decibel hearing loss in the involved ear, if the tinnitus was below 3,000 cycles per second. If the patient had good hearing in the opposite ear, adequate safeguarding of that ear was assured by protecting it with a padded ear phone. All audiometry and noise exposure was

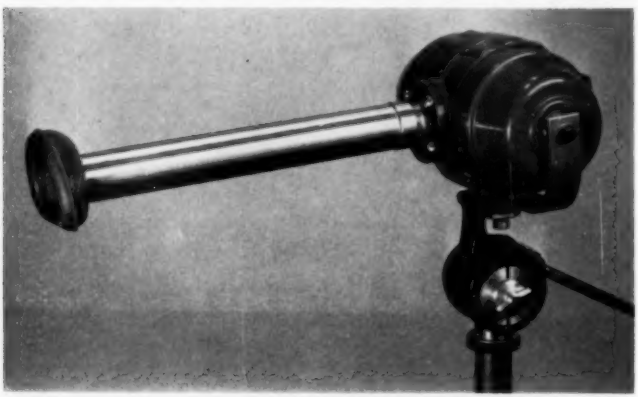

Fig. 2.

done in a sound proof room entirely separated from the technician and the electronic equipment (see Fig. 1).

The traumatizing tone was produced by passing the output of a beat frequency oscillator through a 70-watt amplifier (Scott model 1262-A) to a Jensen 100-watt transducer. The latter is seen in Fig. 2 mounted on a standard heavy-duty camera tripod and equipped with an aluminum tubular extension allowing it to be placed approximately one inch from the patient's ear while he is comfortably seated. The intensity of the noise reaching the patient's ear is measured by a General Radio noise level meter (model 1551-A) connected

with a high-level microphone (Massa Laboratories model M-141-B).

The decibel measurement with this equipment is expressed re: 0.0002 dynes per square centimeter, and all decibel references in our results are on this basis. Before subjecting the patient to the traumatizing tone, the microphone is placed an inch away from the transducer, and the output of the system is then adjusted at the amplifier and/or at the beat frequency oscillator until the desired intensity is reached. Note is made of the dial setting at this point, and the sound is then turned off. The patient takes his place in the inner room, and the intensity is turned up again to the level previously noted.

Following this exposure to the traumatizing tone, the patient is questioned as to the effect on his tinnitus. Analysis of the initial data collected during the first year of study indicated that acoustic therapy seemed to have a beneficial effect. Patients often reported that their ear noises were softer after treatment or had disappeared, but the improvement was seldom more than temporary.

A major question presented itself at this point: could this apparent improvement be a psychological response to the treatment situation? Most patients have been told repeatedly that there is no cure for tinnitus. Despite the fact that acoustic therapy was presented as a matter of research, *i.e.*, that we were trying to find a way to relieve the condition but could not guarantee that this particular treatment would help, many patients apparently regarded it as the answer to their prayers. Few were deterred by having to sign a carefully worded legal release relieving the authors and the hospital from responsibility for any complications or exacerbations of their tinnitus as a result of treatment, or by being told that there was always the possibility that they might be made worse. It is impossible to calculate the psychological implications of this strange new treatment that involved a vast array of equipment and a great deal of preliminary testing; furthermore, tinnitus patients are often described as neurotic and neurotics often tend to be especially suggestible.

With these ideas in mind, we decided to plan a careful

experiment treating 50 patients with tinnitus of cochlear origin and using 50 others as controls. The controls were subjected to an identical procedure, that is, they were given the same battery of tests,* were asked the same questions and were given the same explanation of the treatment with one difference. Instead of being told, "we are going to put into your ear a very loud, piercing, high-pitched sound," they were told "we are going to put into your ear a sound which is actually very loud but which is so high-pitched that it is beyond normal hearing range and, therefore, inaudible." In response to questions about this treatment we would use, if necessary, the term "high-frequency sound" which was meaningless to the patient but which was generally accepted without further comment. We strictly avoided the word "ultrasonic" and when questioned (in two cases) denied that high frequency sound was the same thing.

During the actual "treatment" the control patient sat with his ear an inch from the transducer and was asked not to move his head. After closing the door of the sound proof room, the technician went around to the instrument panel, threw a switch (which actually turned the amplifier *off*) and set a timer for ten minutes. In other words, the "therapeutic atmosphere" of a real treatment was duplicated exactly. Even to a relative or friend who had come with the patient, the whole procedure looked quite authentic. Controls appeared no more dubious and expressed no more curiosity about their treatments than patients who received actual acoustic therapy.

In addition, all patients, treated and controlled alike, were given the Minnesota Multiphasic Personality Inventory ostensibly because we wanted to "learn as much about persons who have this problem as possible." Scores on the Hysteria, Depression, and Hyperchondria scales were used together as an index of neuroticism. A patient was classified as neurotic if two of these three scores exceeded the upper limits of nor-

*See description of the audiometric work-up given each patient in Reed, G. F.: "An Audiometric Study of 200 Cases of Tinnitus," Arch. Otolaryngol., Vol. 71, Page 86.

malcy on the MMPI profile.* A number of patients were eliminated from the study because their inability to read or lack of proficiency in the English language prevented their answering the questions accurately. Two or three persons were eliminated because they refused to take the test.

Patients with tinnitus of cochlear origin were eligible for real acoustic therapy so that the bulk of the controls consisted of those whose tinnitus was of central nervous system, conductive or vascular origin. It was not necessary for controls to be cochlear as the patient himself was never told of this classification or its meaning, and we could see no reason why patients with different types of tinnitus should differ in their degree of suggestibility unless one group tended to be more neurotic than the other. The latter possibility was negated in the matching procedure described below.

There was no difficulty recruiting enough controls so that it was possible to match individually each treated patient to a control on the basis of age, sex, presence or absence of neurotic tendencies, character of tinnitus (constant or intermittent), and duration of tinnitus. In five cases, however, the control was of the opposite sex.

All patients, treated and controlled alike, received three ten-minute treatments approximately a week apart. Each treatment was preceded and followed by an air conduction audiometric test. During the ten-minute period the controls, of course, heard nothing; the treated patients were exposed to a pure tone at an intensity level of at least 120 decibels. The frequency of the traumatizing tone was based in part on the tonal quality of the patient's tinnitus and in part on the maximum intensity obtainable. With the equipment described previously, intensity peaks (maximum 144 decibels) occur at or about 2,000, 3,200, 4,000, 5,800 and 6,200 cycles per second. In practice, the tone used for treatment was the intensity peak closest to, but somewhat above the central frequency of the tinnitus. The hearing of each control was checked at 2,000, 4,000 and 8,000 cycles per second, but that

*The MMPI tests were scored by Miss Christian but final decision as to whether or not a patient showed neurotic tendencies was made by our consulting psychologist, Herbert Terrace, M.A.

of the treated patients was tested at 1,000 cyles steps from an octave below the traumatizing tone through 8,000 cycles per second, in order to record threshold depression and recovery, A patient was not treated a second or third time before, or unless, recovery was complete within the speech range and not unless the threshold had returned to within 10 decibels of the original level in the area above the speech range.

Immediately following each treatment the patient in either group was asked, "Do you now notice any difference in the noise in your (left, right) ear?" If the patient reported that his tinnitus was softer he was asked to approximate the degree of improvement in percentages. His answers and any other remarks pertinent to the effect of the treatment were recorded.

HYPOTHESES.

To summarize the plan of the experiment we have a total of 100 patients divided into two matched groups of 50: group T, those who have been exposed at least three times to a pure tone of 120 or more decibels in intensity for not less than ten minutes at a time; and group C, those who have experienced an identical treatment situation involving three ten-minute sessions during which they were not exposed to any sound. These two groups can be further subdivided into neurotic and non-neurotic on the basis of the Minnesota Multiphasic Personality Inventory results.

We believe that exposure to a pure tone at an intensity level greater than 119 decibels produces a cessation of tinnitus for however short a duration.

There are two null-hypotheses involved: first that such exposure to sound makes the tinnitus worse; second, that such exposure has no effect whatever, or as a corollary, that the treatment situation alone is responsible for the responses seen by virtue of the psychological effect of "being treated." We shall reject the first null-hypothesis if we find no significant difference in the two groups in the number of persons who became worse. We will be unable to reject it, however, if we find a larger proportion of "worse after treatment" cases

among the patients in group T than in group C. We will reject the second null-hypothesis if we find "cessation of tinnitus after treatment" occurring more frequently among the patients in group T than in group C. We will be unable to reject it if no significant difference is found between the two groups, or if a difference in the opposite direction occurs. Assuming further that neurotics are more suggestible than non-neurotics, and, therefore, are more prone to purely psychological responses, we cannot reject the second null-hypothesis if we find that in both groups a significantly greater proportion of neurotics than normals experienced a cessation (or worsening) of tinnitus after treatment.

TABLE I.

Response to Three Treatments.	Treated.		Control.		
	Normal	Neurotic*	Normal	Neurotic	Total
No change after any	4	1	12	6	23
Worse after one treatment	3	3	2	1	9
Worse after two treatments	2	1	1	0	4
Worse after all three	3	1	0	0	4
Some improvement after each	3	3	4	3	13
Gone after one	4	3	1	0	8
Gone after two	6	1	3	0	10
Gone after all three	3	2	0	0	5
Erratic response	5	2	10	7	24
Total	33	17	33	17	100

*As determined by MMPI (see text).

RESULTS.

Table I presents the total picture of the response to treatment on the part of all patients in each of the four possible groups.

The responses listed are mutually exclusive: "Gone (or worse) after one (or two) treatment(s) indicates that the other treatment(s) produced either no change or some slight improvement. If a patient experienced a cessation of tinnitus after one treatment but felt that his noises were worse after another, his "response to treatment" is classified as "erratic." In 13 cases the patient reported that his tinnitus was (25, 50, or 75 per cent) softer following treatment but was never

completely gone. These cases comprise the category "some improvement after each."

In Table II categories have been combined to examine the influence of neuroticism. There is no evidence in these figures to support the contention that neurotic patients differ from non-neurotics in their response to the treatment situation whether or not actual therapy is involved. Table II, of course, shows the total picture, treated and control patients together; however, even if the groups are examined separately no re-

TABLE II.

Influence of Neuroticism as Measured by MMPI—All Patients.

Response to Treatment	Normal	Neurotic	Total
No change after any	16	7	23
Worse after two out of three	6	2	8
Gone after two out of three	12	3	15
Other responses	32	22	54
Total	66	34	100

$X^2 = 3.137$; not significant at the .05 level.

TABLE III.

Differential Effect of Treatment on Treated Patients and Controls.

Response to Treatment	Treated	Control	Total
No change after any	5	18	23
Worse after two out of three	7	1	8
Gone after two out of three	12	3	15
Other responses	26	28	54
Total	50	50	100

$X^2 = 17.322$; significant at the .01 level.

lationship exists between neuroticism and "response to treatment."

According to Table III treated patients differ from controls in that their tinnitus more often disappears after treatment but also more often gets worse. The Chi square test shows a significant deviation from the expected value but does not tell us which result is more likely to occur, getting worse or getting better.

If, in fact, there were no differences between the two

groups, the probability of five treated patients as opposed to 18 controls experiencing no change in their tinnitus as a result of treatment is .005. The possibility of either of the two events "gone" or "worse" occurring 19 times among the treated patients and only four times among the controls is .001. These results indicate that acoustic therapy has an effect on tinnitus which cannot be accounted for by the psychological influence of the treatment situation, since both groups experienced the same situation, but only one was actually treated.

The effect is not necessarily beneficial. Both the probability (.018) of the distribution of the T and C cases in the "gone" category (12 to 3) *and* the probability of the distribution in the "worse" category (.062) are very small. Since exposure to sound is the only factor distinguishing the two groups, acoustic therapy has to account for *both* these results. We can reject the null-hypothesis that acoustic therapy has no effect on tinnitus, but it is not possible to predict which effect it will have.

However significant these results may be statistically, clinically the number of treated patients who were helped by the treatments is small. A number of factors were examined to see what, if any, difference existed between the patients whose tinnitus was gone after at least two out of three treatments and those who got worse or experienced no change. No correlation was found between the response to treatment and any of the following: age, character (intermittent or constant), duration or central frequency of tinnitus, type of hearing loss, previous exposure to trauma, frequency or intensity of the traumatizing tone used in treatment.

Table IV shows the relationship between threshold shift, that is, the amount of further hearing loss sustained following *each* treatment and the response to that treatment.

Although this distribution is significant it is not possible to relate the magnitude of the shift to any specific response category. There is some indication that the patients whose threshold shift was greater than thirty decibels more often

fell into the "worse after treatment" category. It is not possible to show increasing degree of improvement with increasing *or* with decreasing threshold shift. The significant distribution in Table IV may be spurious, and in any case does not enable us to distinguish reliably between those cases in which tinnitus was worse after treatment and those which were gone.

To summarize these findings, significantly more treated than control patients were affected by treatment; but the effect goes in both directions, and it was not possible to pin-

TABLE IV.

Relationship of Maximum Threshold Shift and Response to Treatment; Treated Cases, All Treatments.

Response to Treatment	0-10 db.	11-20 db.	21-30 db.	31 db. and up	?*	Total
Worse	1	8	6	7	4	26
No Change	14	17	11	3	0	45
Some Improvement	21	7	8	2	4	42
Gone	10	12	5	4	6	37
Total	46	44	30	16	14	150

*This category represents those people whose hearing threshold was greater than 100 decibels so that the shift following treatment could not be measured. When this group is added to those in the category 31 and up (which is not necessarily logical and the actual shift involved might be considerably less depending on the original threshold), the distribution is significant at the .01 level; $X^2=28.305$.

point any factor(s) which distinguish between the "gone after treatment" and the "worse after treatment" cases. In view of this situation we are again led to consider the possibility of a psychological explanation. The effect of the treatment situation was controlled, so it is not the fact of being treated that accounts for the results noted; nevertheless, it is not possible to control for the psychological influence on the patient exposed to sound of such great intensity. If acoustic therapy is actually affecting tinnitus on a psychological rather than physiological level, the occurrence of two opposite responses, worse and gone, is more understandable.

SUMMARY.

A study of the effects of loud sound on tinnitus was made,

using a sample of 50 treated people matched individually with 50 controls on the basis of age, sex, duration and character of tinnitus, and presence or absence of neurotic traits as measured by the Minnesota Multiphasic Personality Inventory. Each treated patient received three consecutive treatments at least a week apart consisting of a ten-minute exposure to a pure tone of an intensity greater than 119 decibels. The controls experienced an identical therapy situation before, during and after what they understood to be a ten-minute "treatment" with the one difference that they were not exposed to applied sound.

The results indicate that such exposure to a traumatizing tone may have a two-sided effect: either to alleviate the tinnitus or to increase its severity. In either case the effect is only temporary; however, because the effect of acoustic therapy is not necessarily beneficial it is not possible with this data to substantiate the hypothesis that further partial destruction or fatigue of the cochlea relieves tinnitus even temporarily. While it is true that the effects of this form of therapy cannot be accounted for by the psychological implications of "being treated" or by simple suggestibility, it is still possible to explain the results noted in terms of the psychological influence of exposure to very loud sound on the patient. The role of this factor is a moot point in the opinion of the authors, as there seems to be no way to control for it.

It was incidentally demonstrated that neurotic tinnitus patients are no more or less suggestible than non-neurotics; furthermore, using as a measure the hypochondria, depression and hysteria scales of the MMPI, the proportion of neurotics (34 per cent) found in this sample of 100 tinnitus patients is far below the 70-80 per cent reported by Fowler and others on the basis of clinical observation.

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STAPEDIAL FRACTURE FOLLOWING HEAD TRAUMA.

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With the advent of microsurgical techniques in otology, deviations from the usual pathological findings are now appearing more frequently; therefore, it becomes necessary for the clinician to be aware of the more unusual situations that may occur with a conductive hearing loss. For this reason I am presenting this paper, and also adding another case report to the accumulating literature showing that head trauma can result in ossicular injury with a restorable hearing loss.

Ossicular injury has been known to occur after head injury with varying degrees of conductive and/or sensori-neural hearing loss. Until recently, most head injuries resulting in ossicular damage were usually severe enough to be fatal, and the pathological descriptions were usually reported on a post-mortem basis. With the advances and improvements in the care of head injuries, more patients now survive and recover, only to find that there is resultant damage to the auditory apparatus.

Several excellent discussions and papers have described the role of head injury and auditory damage. Schuknecht[1] describes subluxation of the incudostapedial joint in severe cases of longitudinal fracture of the temporal bone. A more recent well documented report on incudostapedial joint separation and treatment was presented by J. V. D. Hough[2] in 1959 with case reports. Thorburn[3] in 1957 reported a case of bilateral conduction deafness due to incudostapedial joint subluxation and performed bilateral Type III tympanoplasties with good improvement in hearing. Gisselsson[4] in Sweden, described a case with head injury and at exploration found a disarticulated incus. In one ear he reduced the dislocation and in the other ear he created a columella effect by inserting the body

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of the incus between the stapes and the drum, with almost complete closure of the air-bone gap. Hough[2] summarizes very well the factors which may produce the incudostapedial joint separation in head trauma: *1.* the jarring effect, producing derangement of all moveable parts according to the physical laws of inertia; *2.* possibly a sudden severe tetanic contraction of the tympanic muscles with an abrupt change in the axis of rotation of the ossicles.

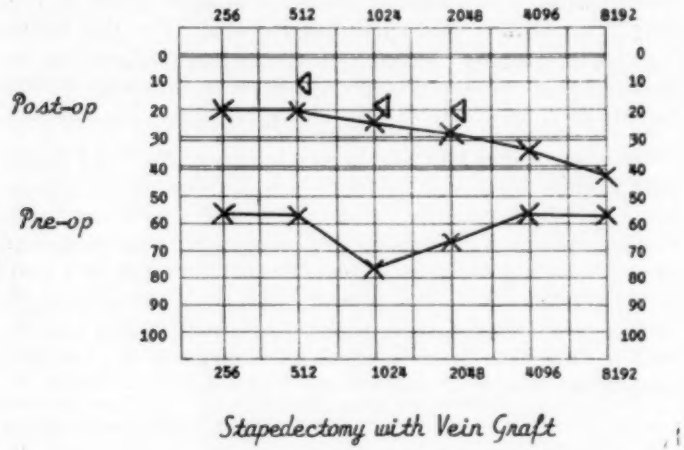

Fig. 1.

This paper presents another type of ossicular injury in addition to the incudostapedial joint subluxation. The following case illustrates a fracture of the stapes as well as a subluxation of the joint, and a method for successfully treating this condition. Review of the literature fails to reveal any reported cases of a fractured stapes; but, no doubt, these will appear more frequently as otologic microsurgery is utilized to investigate all suspicious conductive hearing losses following head injury.

The following case is that of a 40-year-old white female, who, in May, 1959, sustained a head injury as a result of a small foreign sedan turning over at high speed. The patient was thrown from the automobile.

No skull fracture was evident by X-rays, but she did have a period of unconsciousness for several minutes. She also sustained a fracture of the fifth lumbar vertebra. Previous to this episode she stated that her hearing was normal, but following the return of consciousness she noted that voices appeared distant. There was slight vertigo and loss of balance for several weeks after the injury, but no tinnitus at that time. Following the injury she developed a blocked sensation in the left ear suggesting a hearing loss. She was seen five months after the injury for otological examination and evaluation of her hearing loss.

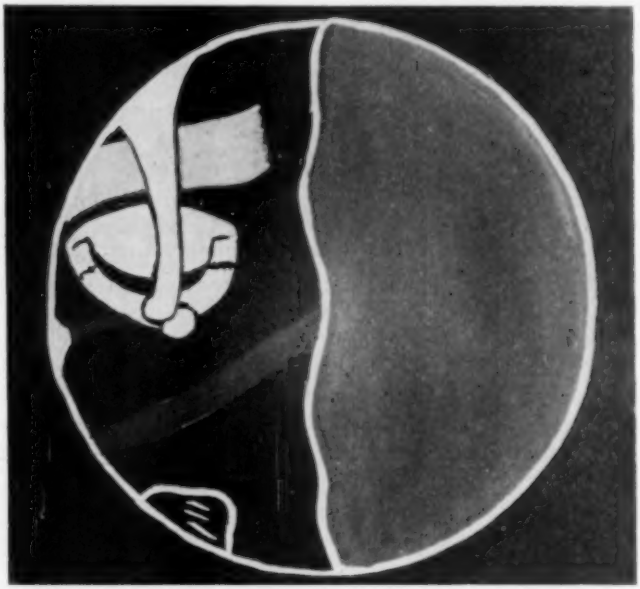

Fig. 2.

Examination revealed both ear canals to be normal without evidence of trauma. The tympanic membranes also had a normal appearance and excellent movement with the pneumatic otoscope. No nystagmus was present, and her neurological test was also normal. Audiometric tests revealed a 30 decibel sensori-neural type hearing loss in the right ear, and a 65 decibel conductive type hearing loss in the left ear (see Fig. 1). Caloric vestibular tests revealed normal labyrinthine function. Initial impression following this evaluation was a disarticulation of the incudostapedial joint in the left ear secondary to head trauma, and a mild perceptive hearing loss in the right ear as a result of cochlea concussion.

Eleven months following the head injury an exploratory left tympanotomy was performed, using the operating microscope. Immediately evident was the incudostapedial joint, where the head of the stapes was noted

to be subluxated from the lenticular process of the incus and displaced 1 mm. inferiorly (see Fig. 2). Palpation of the tympanic membrane revealed movement of the incus, but no transmission of movement to the stapes. Further investigation of the stapes under higher magnification revealed a fracture of the crura at the crural-footplate junction. The footplate itself was moveable and attached to the crura by mucous membrane. Because of the fracture-dislocation of the stapes, it was decided to insert a polyethylene prosthesis and attach it to the incus. The crura were removed, but because of poor positioning of the beveled edge of the polyethylene prosthesis it was decided to remove the footplate and insert a vein graft. This was performed without difficulty, and the polyethylene prosthesis was attached as in the usual Shea stapedectomy. The tympanic membrane was replaced in its original position, and the patient's hearing improved immediately.

Her postoperative course was uneventful, and her hearing improved from the 65 decibel level in the speech range to the 25 decibel level, actually producing better hearing in the operated ear (see Fig. 1).

SUMMARY.

Ossicular injuries are infrequently reported, but the increased use of the operating microscope for conductive hearing losses will undoubtedly result in more findings similar to those described above. Although incudostapedial joint separation has been described and its treatment outlined, no similar papers have been published in regard to stapedial fracture as a direct result of head trauma. This paper should reinforce the principle that not only is a good history necessary when an individual is found to have a conductive type hearing loss, but also that exploratory tympanotomy is an indicated procedure and that reconstruction of the ossicular chain by direct repair or substitution can be accomplished successfully.

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MALIGNANT MELANOMA OF THE LARYNX.*

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The rare occurrence of a primary malignant melanoma in the larynx merits a report of a case and a review of the pertinent literature.

Approximately 98 per cent of the total tumors are of squamous cell origin and characterized as epidermoid carcinoma of various degrees of differentiation. About 1 per cent are of glandular origin and appear as adenocarcinomas or cylindromas. The remaining 0.5 per cent encompass the relatively rare sarcomas of mesenchymal tissue derivation.[6-11] In this latter uncommon group the malignant melanoma is the medical oddity.

CASE REPORT.

A 41-year-old white female was examined for hoarseness of four months' duration and recurrent bouts of pneumonia. Constant left parietal headache had been present for eight months, becoming progressively worse and partially relieved by salicylates. Gradual weight loss was associated with anorexia and intermittent febrile episodes. A chronic non-productive cough with pleuritic pain was increasing in severity. Examination revealed a first degree horizontal nystagmus on right lateral gaze. The nasopharynx was filled with a brownish polypoid mass in the middle of the vault. The laryngeal motility on the right showed limitation of motion about the paramedian position. There was an irregular tumor mass on the posterior third of the right vocal cord. Palpation of the right deep cervical chain disclosed small discrete nodes extending below the clavicle. Audiometric studies demonstrated normal hearing in the right ear and a 60 db inner ear loss on the left side.

Chest roentgenograms on admission outlined a right lower lobe atelectasis with mediastinal shift, and seven days later showed a further increase in the atelectasis with pleural effusion. Films of the mastoids, petrous apices, and basilar view of the skull were interpreted as normal.

Lumbar puncture was performed and the CSF protein was elevated to 89 mgm. o/o (normal 12-50 mgm. o/o). The cell count was less than

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10/mm.^{3} Quantitative analysis of the urine for melanin was reported within the normal range.

Biopsy of the nasopharyngeal and laryngeal masses was carried out and the tissue submitted for histologic study. The photomicrographs show representative sections from the respective areas (see Figs. 1 and 2).

In addition to the proven neoplastic areas, the steady progression of intrathoracic signs, *i.e.*, effusion, atelectasis, and repeated bouts of pneumonia, represent strong presumptive evidence of widespread metastatic activity of the melanoma. Probable posterior fossa extension from the

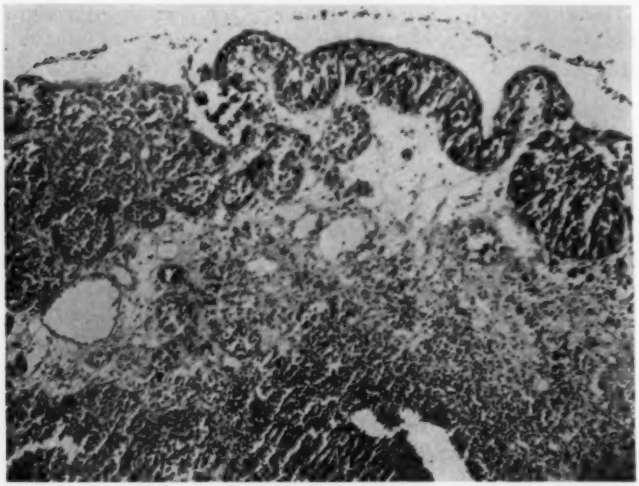

Fig. 1. Nasopharynx: In some areas the respiratory epithelium of the nasopharynx demonstrated an apparent thickening of the tunica mucosa due to proliferating nevus cells situated above the basement membrane. Immediately adjacent to this zone of junctional activity, at the left on the illustration, the mucosa is deficient, and malignant nevus cells extend deeply into the submucosa in characteristic clusters.

nasopharynx was suggested by the persistent cephalgia, nystagmus, and recent unilateral left hearing loss of a perceptive type.

The widespread activity of the neoplasm precluded further diagnostic and therapeutic methods; for this reason the patient was discharged from the hospital with analgesic medication.

DISCUSSION.

Malignant melanoma occurs much less frequently in mucous membranes than on cutaneous tissues, and its presence in the upper respiratory tract accounts for about 10 per

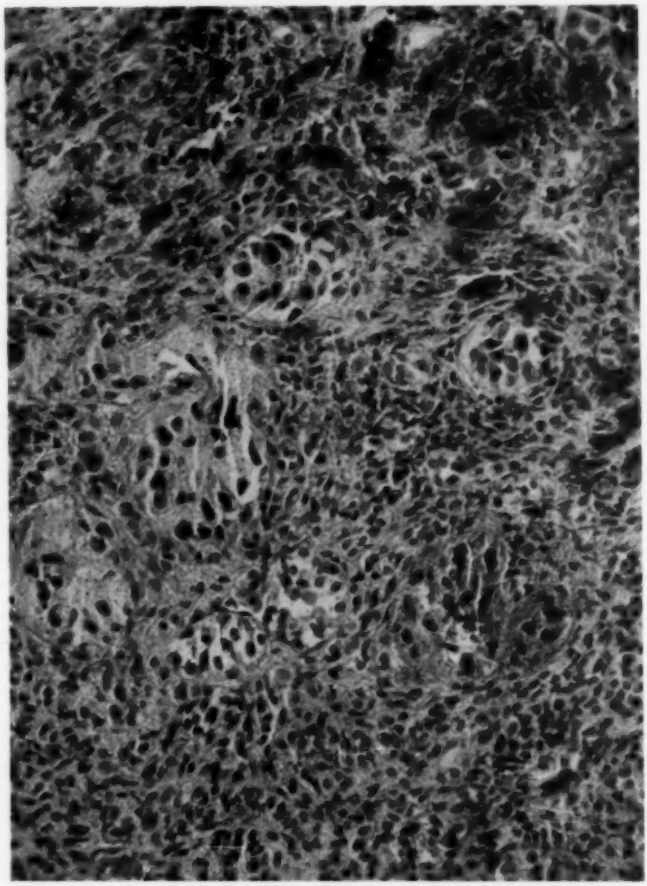

Fig. 2. Larynx: The higher magnification of the laryngeal neoplasm shows the gradation in differentiation of the melanoma from the easily recognized pigment-containing cells, which are cytologically fairly uniform (top), to the undifferentiated tumor (bottom). The central area of the photomicrograph contains more pleomorphic cells which have elaborated relatively little melanin. The upper two zones are histologically identical with those seen in various areas of the nasopharyngeal specimen.

cent of the total number. The area is usually pigmented, though a small percentage are without color and present as a dense mass of newly developing tissue. Epistaxis, fullness, obstruction and metastases are the common symptoms which encourage examination. In contrast to the late diagnosis associated with most melanomas in this region, hoarseness of recent onset indicates relatively short duration of the disease.

There is sufficient evidence to support the clinical observation that primary melanocarcinoma in the mucous membranes offers a poorer prognosis than cutaneous and genital lesions, due to the presence of relatively more mitotic figures and pleomorphism.[1] In addition, the presence of this neoplasm may activate nearby junctional nevi into overt carcinomatous activity.

We are unable to state with certainty in this case whether both sites are primary areas of neoplastic activity. The histologic characteristics of the nasopharyngeal specimen clearly show the accepted sequence of melanoma derivation, from intraepithelial malignancy to frank invasion which is well recognized on skin surfaces. In the laryngeal specimen, we were unable to demonstrate this order in the available slides; however, the alternatives, *i.e.*, lymphogenous spread or seeding to the larynx, are so unlikely that we prefer to consider it a second primary tumor.

A summary of available cases in the literature is presented to indicate the mode of onset, clinical appearance, therapy, and the eventual outcome when follow-up data were recorded (see Table I).

The first recorded instance of malignant melanoma of the larynx was described by Hilterman in 1908. The case was not further discussed, and hence the outcome is not available. On the initial examination the patient was observed to have a black-bluish neoplasm on the laryngeal aspect of the left epiglottis with enlarged right cervical nodes.

It is fairly obvious from the above reports that the prognosis is not encouraging. Radiotherapy may be considered, but an important fact is the marked insensitivity of the nevus

TABLE I.

Author	Description	Therapy	Follow-Up
1. Havens and Parkhill	Dark brownish mass on the right arytenoid cartilage and hypopharynx.	None	Widespread metastases on examination
2. Moore and Martin	Bulky reddish polypoid tumor mass arising from the left ventricle.	Laryngectomy	Deceased—14 months
3. Moore and Martin	Same findings.	Laryngectomy	Deceased—21 months
4. Curtiss and Kosinski	Smooth, thickened, slightly enlarged right arytenoid.	Radiotherapy	Deceased
5. De Juan	Black-bluish mass arising from left epiglottis, laryngeal surface.	Laryngectomy and radical neck dissection	Unknown
6. Cremonesi	Pinkish gray mass with brownish hue on the posterior third of left true cord invading the ventricle.	Radiotherapy and laryngectomy	Unknown
7. Welsh and Welsh	Raised pigmented area on arytenoid. Pigmented polypoid tissue in nasopharynx.	None	Diffuse metastases on examination

cell, offering as much as and often more resistance to radiation than the adjacent tissue. Significant response to radiotherapy is noted in only 3 to 4 per cent of cases. Chemotherapy has been resorted to as mentioned by Allen and Spitz when they refer to Farber's use of triethylenephosphoramide (TEPA) on a large scalp lesion which underwent marked regression.

Conclusions regarding therapy can hardly be drawn from a small series such as this review presents. Whenever clinically evident metastases are present, as in this case, chemotherapy and/or surgery are generally recognized to be of little value.

SUMMARY.

The fact that malignant melanoma may exist simultaneously in several primary sites has been illustrated in the nasopharynx and larynx. The late onset of symptoms and the evidence of widespread metastases in the reported cases indicate the poor prognosis of this lesion. A review of reported cases reveals that all five of the determinate cases are deceased or pre-terminal from metastases.

ACKNOWLEDGMENT.

The authors appreciate the contribution of Dr. George Zimmerman in reviewing and commenting upon the slides.

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PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The next annual meeting of the Pennsylvania Academy of Ophthalmology and Otolaryngology will be held May 18-19-20, 1961, at the Bedford Springs Hotel, Bedford, Pa.

The next annual meeting of the American Society of Ophthalmologic and Otolaryngologic Allergy will be held Saturday, October 7, 1961, at the Sherman Hotel, Chicago, Ill. The morning program will consist of a workshop on Diagnosis and Management of Allergic Disease. The afternoon program will consist of scientific presentations on Allergy, Immunity and Medical Otolaryngology.

The Twelfth Annual Eye and Ear Hospital of Pittsburgh Scientific Day will be Wednesday, April 19, 1961, at the Eye and Ear Hospital, Pittsburgh, Pa. The morning program will consist of a workshop on rhinoplasty and nasal and sinus diseases. The afternoon program will consist of scientific presentations in Ophthalmology and Otolaryngology.

ESTHESIONEUROEPITHELIOMA.

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and
AUBREY K. BROWN, Lt. Col., MC, USA*
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Three per cent of all intranasal tumors, excluding nasal polyps, are primary neurogenic tumors. Of these, the esthesioneuroepithelioma is the most uncommon. To date, only 31 have been described in the world's literature. Two additional cases are reported in this paper.

The esthesioneuroepithelioma was first described by Berger and Luc[1] in 1924. They reported a single case which occurred in a 50-year-old man. In 1926, Berger and Coutard[2] reported a second case which was diagnosed as an esthesioneuroepithelioma olfactif because it possessed a somewhat different microscopic picture. Twelve additional cases were reported in the European literature before the first American case was reported in 1951.[3] The rarity of these tumors could be the result of the confusing pathological picture which is seen.

In his discussion on neuroblastomas of the nasal cavity, Fisher[4] classified intranasal neurogenic tumors as developmental, and neoplastic. The developmental tumors include the nasal glioma, encephalocele, and basal hernia. The neoplastic tumors are the neurofibromata, the neurilemmomata, and the ganglioneuromata.

The five theories of histogenesis center around the abundance of neuro-epithelial tissue in the nose.

1. Olfactory membrane: This membrane is made up of two cell types, neuroepithelial and olfactory epithelial. The supporting cells and the neurocytes of the olfactory membrane originate from the same stem cell, the esthesioneuroblast.

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This cell may differentiate into two types of cells, producing the esthesioneurocytoma. These two cell types are the basis for differentiation of the two tumor types described below.

2. The Organ of Jacobson: Ordinarily vestigial, this structure is occasionally seen in the adult. It is located in the antero-inferior area of the nasal septum. This is an unlikely choice as a primary site of origin.

3. The Sphenopalatine Ganglion: The location of this ganglion outside of the nasal cavity makes it an unlikely site of origin. Mendeloff stated that it would be difficult to explain the rosette formation of Pattern II, with a tumor of sympathetic neuroepithelial origin.

4. The Olfactory Placode: Since this structure does not exist beyond the embryonic state, its selection is also unlikely.

5. Ganglion of Loci: Martin, Dargent and Gignoux[5] assumed that some of these tumors arose from this site which is at the anterior end of the olfactory placode. Its pathways to the brain are not well described. It belongs to the nervus terminalis.

This histopathology of the olfactory esthesioneuroepithelioma is similar to that seen in tumors of the adrenal medulla, the ganglia of the sympathetic nervous system, and the retinoblastoma. They are divided into two histologic patterns:

Pattern I: Termed the olfactory neurocytoma, has sheets and cords of undifferentiated cells separated by connective tissue septa. Occasional pseudorosettes are seen, as well as some neurofibrils. Mitotic figures are rarely seen. Only Pattern I is reported to metastasize.

Pattern II: Termed the neuroepithelioma, this type demonstrates true rosettes which are lined with columnar epithelium. Areas which are more typical of Pattern I may be found interspersed between fields of rosettes. Pattern II occurs less frequently than Pattern I.

Sprinz[6] does not use this classification. He considers the degree of differentiation to be the most logical determinant of metastatic activity.

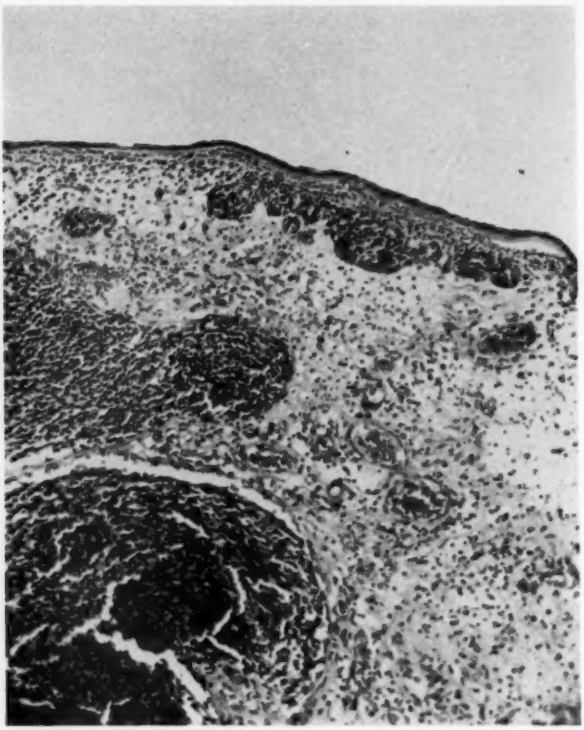

Fig. 1. Undifferentiated cells separated by connective tissue septa characterized the histopathology of the tumor.

Fisher designated the tumors as neuroepithelioma when rosettes were present, and neuroblastoma when pseudorosettes and abundant fibrils were found.

Local invasion into the ethmoid, maxillary, and frontal sinuses as well as invasion into the orbit and cranial cavity can occur. Distant metastases have been described only in patients with Pattern I tumors.

The first symptoms of this tumor are nasal obstruction and epistaxis. The physical examination may reveal a polyp-like mass filling the nasal fossa, reddish in color, and firm to pal-

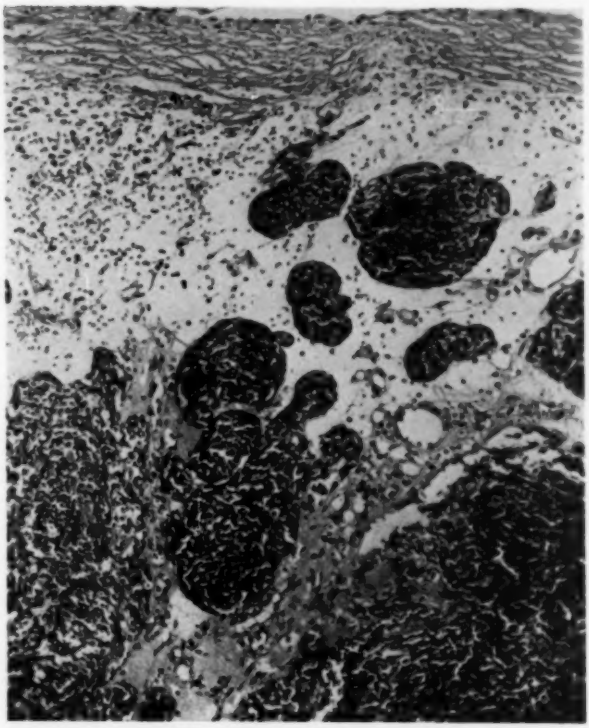

Fig. 2. An undifferentiated form of esthesioneuroepithelioma with sheets and cords of undifferentiated cells.

pation. It is usually found high on the lateral nasal wall in the area of the ethmoid labyrinth. X-rays of the sinuses may show only clouding of the ethmoid area, or evidence of a mass with bony erosion might be present. Calcification may be seen. Fig. 1 shows a whorled calcification of the left antrum and ethmoid area as found in Case 2.

The differential diagnosis includes nasal polyps, adenocarcinoma, sarcoma, undifferentiated epithelial carcinoma and malignant melanoma.

Mendeloff[7] reviewed the world's literature and added six

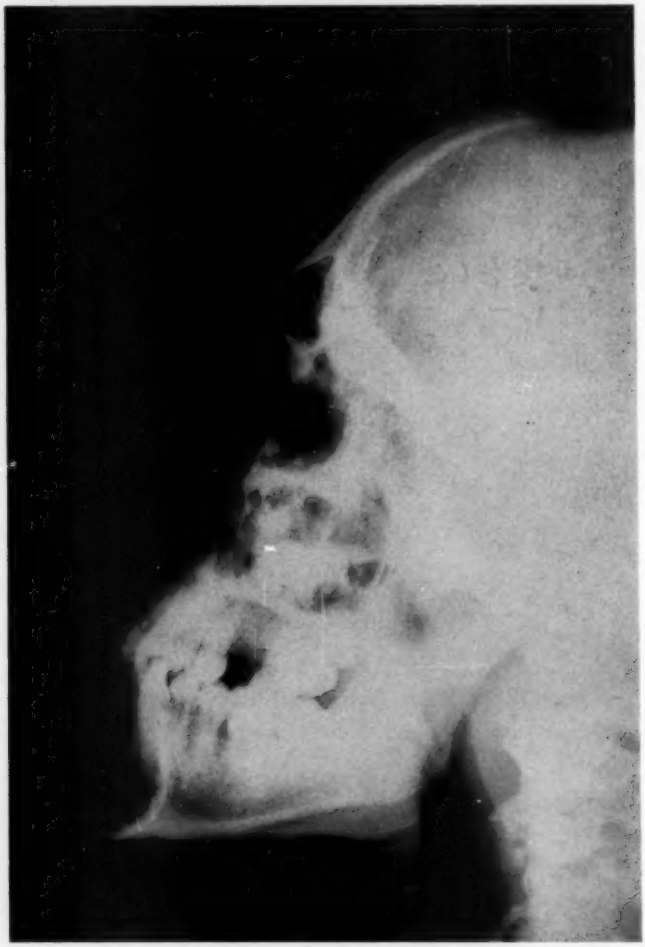

Fig. 3. The lateral projection shows the calcified tumor mass involving the left antrum, and the ethmoid area.

cases of his own. He found the sex distribution to be 14 males and 14 females. One of our cases was an 18-year-old boy, the other a 36-year-old woman.

Treatment consists of surgery and radiation. Both types are extremely radiosensitive. Fruhling and Wild[6] stated that radiation in moderate doses gives spectacular results. They supported the combination of surgery and radiation.

The prognosis should be guarded, regardless of type, because of the possibility of recurrence or metastases. Mendeloff reported single or multiple recurrences in six patients. Seaman reported one case with two recurrences.

Case 1. In this 36-year-old woman, initial symptoms began in July, 1952. At that time she began to have recurrent nose bleeds. She did not seek medical attention until September, 1953. Examination at that time revealed a necrotic polyp in the left nasal fossa. A biopsy was performed, and the report was returned as undifferentiated carcinoma. The patient was transferred to Walter Reed General Hospital at that time but refused further treatment. She felt well until October, 1956, when she was admitted to another hospital complaining of nasal obstruction. Examination revealed the left nasal fossa to be filled with granulation-like tissue. A left ethmoidectomy was then performed, and the tumor was found to be invading the cribiform plate. The pathologist again reported an undifferentiated carcinoma. She was re-admitted to Walter Reed General Hospital in January, 1957. Review of the tumor material resulted in a diagnosis of olfactory esthesioneuroepithelioma (see Fig. 1).

On February 1, 1957, the patient was operated upon under general anesthesia, and a radium needle implant was inserted in the area of the ethmoid sinuses. Fourteen radium needles were implanted. A total of 24.3 mg. of radium was used, and left in place 117 hours. A total of 6,494 gamma roentgens was received in the tumor-bearing area.

In addition, the patient received external radiation through an anterior head port with a tumor dose of 3,200 *r* from February 7, 1957, to February 22, 1957. She was discharged from Walter Reed General Hospital on March 5, 1957, and has remained tumor-free since that time.

Case 2. This 18-year-old Caucasian boy began having frontal headaches in February, 1958. He then noticed gradually increasing left-sided nasal obstruction. He reported to his hospital facility where the diagnosis of nasal polyp was made. Excision was performed as a clinic procedure and resulted in profuse hemorrhage. A left lateral rhinotomy and external carotid ligation were performed. A tumor involving the left ethmoid and maxillary sinuses was found. A Caldwell-Luc procedure was performed in November, 1958, because residual tumor was thought to be present. The Armed Forces Institute of Pathology classified the tumor as an esthesioneuroepithelioma (see Fig. 2). The patient was then transferred to Walter Reed General Hospital.

The admission physical examination revealed a left lateral rhinotomy scar, with intranasal evidence of surgical removal of the left inferior and middle turbinates. A 0.5 cm. area of residual tumor, flesh-colored, and mulberry-shaped was seen high in the ethmoid area.

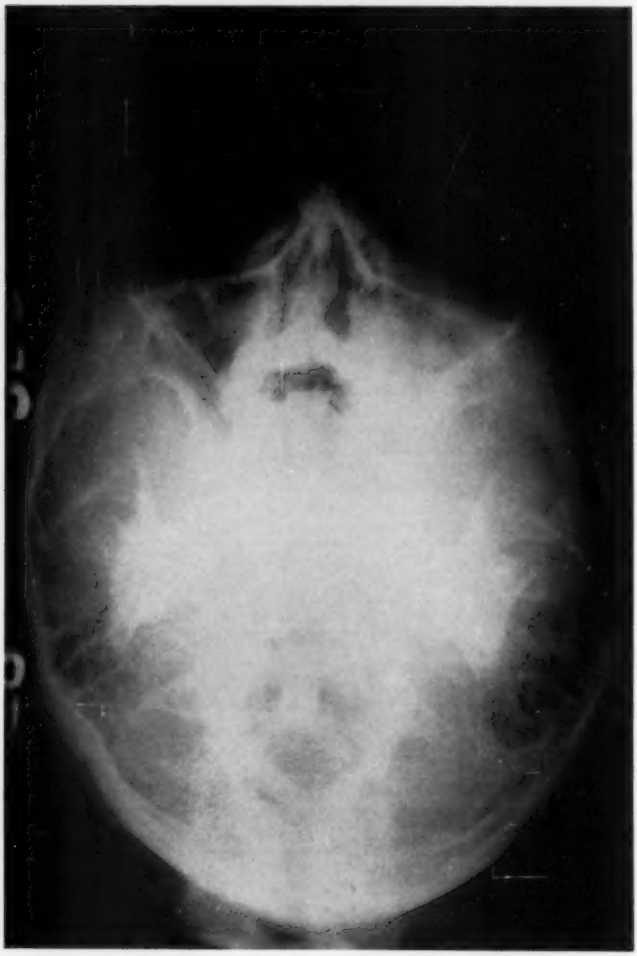

Fig. 4. The Waters projection reveals a calcified tumor mass in the left antrum.

Review of the original X-rays showed a whorled calcific tumor mass involving the left antrum and ethmoid areas (see Figs. 3 and 4).

The residual tumor in the ethmoid area was removed under general anesthesia. On March 30, 1959, 12 radium needles were inserted into the tumor-bearing area. A total of 18.0 Gm. of radium was used and left in place 143 hours. A total of 7,000 gamma roentgens was delivered to the tumor-bearing area. A right inferior rectus paresis developed which gradually resolved. The patient was last seen in December, 1959, free of tumor.

SUMMARY.

1. The esthesioneuroepithelioma is an extremely rare tumor.

2. The most likely origin is the olfactory membrane.

3. The clinical history is briefly discussed, and two additional cases are described.

4. Treatment consists of a combination of radiation therapy and surgery.

5. The prognosis is guarded.

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PREOPERATIVE DETECTION OF BLEEDING DISORDERS.*

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For a long time the detection of latent hemorrhagic disorders has been an important and controversial problem in surgery. Many surgeons have experienced sad and sometimes tragic surprises during and after operations with severe hemorrhages due to unexpected disorders in the clotting mechanism; consequently, surgeons have always been very anxious to find a foolproof procedure to detect such conditions.

Congenital bleeding disorders in adults rarely surprise the surgeon, since injuries in childhood will draw attention to the abnormal blood coagulation. Even in adults, the very first operative procedure may uncover an unexpected bleeding tendency of a congenital, or, more frequently, of an acquired and transient nature. The most distressing accidents of this type, however, occur in early childhood; consequently, investigators studied this problem in children before and after the first surgical intervention, *e.g.*, tonsillectomy. Frequently the investigations were done after a serious hemorrhage following an operation.

Several papers dealt with the medicolegal and hematological aspects of this problem. Two articles[1,2] recommended the following procedure to detect bleeding disorders:

1. It is generally accepted that the commonly performed clotting and bleeding time tests are less than valueless, because they are crude and inaccurate methods, giving a false sense

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of security to the surgeon or causing unnecessary alarm when no abnormality exists. These tests should be discarded.

2. A careful clinical and family history regarding bleeding disorders must be taken.

3. When a history of bleeding is obtained, a battery of special tests should be performed and interpreted by experts to determine the nature of the deficiency.

We fully agree with the first statement. The second is an important suggestion. The third recommendation is theoretically sound but impracticable. Operations on children, *e.g.*, tonsillectomies, are performed frequently in all hospitals. Experts in blood coagulation are few and even fewer are the hospitals that can afford specially trained personnel and equipped laboratories for such investigations; consequently, many surgeons perform operations without any preoperative tests for the detection of bleeding disorders. Empirical observations have taught the surgeons that blood transfusions will stop any kind of bleeding. Since blood is available in every hospital, this became a universal cure for hemorrhagic accidents; however, it is well known that transfusion does not stop bleeding in every defect of blood coagulation, and it is not devoid of dangers.

During the past two years we studied this problem in 400 children (394 cases of tonsillectomy) admitted to the Kingston General Hospital for surgery.

METHODS.

We performed preoperatively the following tests on each patient:

1. Standard Clotting Time (SCT).

Six years ago we developed a highly reproducible test for measuring the overall coagulability of whole venous blood.[3] This test is simple and does not require reagents or expensive equipment. It can be learned quickly by any intelligent nurse or technician; however, it must be performed with meticulous

care. This test gives highly reproducible readings, the standard error of technique being ±0.13 min. The normal values in healthy male adults are 11.9 ±0.9 min.

2. Qualitative clot retraction and/or platelet count.

3. Capillary fragility test with Petechiometer.

When one of these tests was abnormal, special tests such as prothrombin time, prothrombin consumption, thromboplastin generation test and determination of circulating anticoagulants were performed in cases where the parents or patient accepted such extended examinations.

TABLE I.

Laboratory Tests and Operative Hemorrhagic Accidents in 400 Children.

Group	No. of Patients	SCT Min.	Platelet Count or Clot Retraction	Capillary Fragility	Special Tests	Operation	Hemorrhage
A	348	11 - 15½	Normal	Normal	—	Yes	4 Mild (1.1%)
B	43	16 - 18	Normal	Normal	Normal in 11 Cases	Yes	4 Mild 3 Severe 16.2%)
C	6	18½ - 36	Poor	Normal	Abnormal	Deferred	—
D	3	11 - 15½	Normal	Increased	—	Yes	1 Severe

RESULTS.

1. We found in 348 out of 400 children (Group A) between the ages of two and 14 years a Standard Clotting Time of 11 to 15½ min., the average being 13.4 ±0.9 min. (see Table I). All these children had normal capillary fragility and platelet count or clot retraction. Among these patients we observed four who had minor hemorrhage (1.1 per cent) during tonsillectomy, not necessitating any special treatment.

2. In Group B (43 children) with a Standard Clotting Time of 16 to 18 min. we observed seven bleedings, three necessitating transfusions. Capillary fragility, platelet count or clot retraction was normal. In 11 cases special tests gave normal results. At the beginning of our study we did not know what to do with the patients of Group B. Since special tests in the

first cases gave normal results, we permitted tonsillectomy; however, the fact that the incidence of bleeding was 16.2 per cent shows that the hemostasis was not completely normal. Later we adopted the policy of typing and cross-matching blood for possible transfusion in such cases and of keeping the child in the hospital for two or three days after surgery.

3. In six cases (Group C) the Standard Clotting Time was between 18½ and 36 min. Special tests revealed two cases of hemophilia, two Christmas factor deficiency, one hypoprothrombinemia and one case of circulating anticoagulants. The diagnosis of a serious bleeding disorder was made at bedside. The special tests determined the exact nature of the disorder several days later. Surgery was deferred in all cases.

TABLE II.

Laboratory Tests in 29 Children with History of Bleeding.

No. of Cases	SCT Min.	Clot Retraction	Platelet	Capillary Fragility	Special Tests	Operation	Hemorrhage
3	16 - 18	Normal	Normal	Normal	Normal	Yes	None
4	18½ - 36	Poor	Normal	Normal	Abnormal	Deferred	—
22	11 - 15½	Normal	Normal	Normal	—	Yes	None

4. In three cases (Group D) the Standard Clotting Time and clot retraction were normal, but capillary fragility was increased and one of these patients bled during tonsillectomy.

5. An important facet of our study was the finding that 29 of the 400 parents gave some history of bleeding in the child or amongst the relatives (see Table II). In this group three had a Standard Clotting Time of 16 to 18 min. (moderately prolonged) but had uneventful operation. Four had markedly prolonged Standard Clotting Time, abnormal special tests showing a major defect in blood coagulation; therefore, operation was deferred. Twenty-two had normal Standard Clotting Time, clot retraction, platelet count, capillary fragility, and no hemorrhage occurred after tonsillectomy.

6. In the group of 371 children *who did not have* any history of bleeding (see Table III) we found two cases with markedly prolonged Standard Clotting Time demonstrating major coagu-

lation defect (one Christmas factor deficiency and one hypoprothrombinemia), and operation was deferred. In both cases special tests showed the abnormality several hours or days later. In 40 children with moderately prolonged Standard Clotting Time we could not elicit a history of previous bleeding in them or their families, even on repeated questioning of parents. In eight cases special tests gave normal results. Seven children of this group bled during or after tonsillectomy.

DISCUSSION.

Hemorrhagic accidents have various etiology during and after surgical interventions. To understand the significance

TABLE III.

Laboratory Tests in 371 Children Without a History of Bleeding.

No. of Cases	SCT Min.	Clot Retraction	Platelet	Capillary Fragility	Special Tests	Operation	Hemorrhage
2	18½ - 36	Poor	Normal	Normal	Abnormal	Deferred	—
40	16 - 18	Normal	Normal	Normal	Normal in 8 Cases	Yes	3 Severe 4 Mild
3	11 - 15½	Normal	Normal	Increased	—	Yes	1 Severe
326	11 - 15½	Normal	Normal	Normal	—	Yes	4 Mild

of our results we have to discuss the mechanism of hemostasis in a practical and clear terminology.

1. The formation of a perfect clot is one of the most important factors in stopping bleeding. This necessitates a proper interaction of all the clotting factors of the circulating blood. Any defect in this mechanism may produce excessive bleeding.

2. The capillary system itself participates in the hemostasis. After injury of the wall of a capillary, a potent vasoconstrictor serotonin is released from the platelets. This vasoconstriction reduces the lumen of capillaries to such an extent that the blood flow stops completely in them. In the absence of platelets this traumatic constriction of capillaries does not occur;

furthermore, the disintegrating platelets release several substances which participate in the first phase of blood coagulation. Thrombocytopenia consequently can maintain a prolonged capillary bleeding through the absence of capillary contraction and a deficient clot formation. There is another obscure abnormality of the capillaries: the increased fragility seen, for example, in ascorbic acid deficiency which may cause oozing.

3. Bleeding from the larger vessels ceases if the vessel is properly ligated, if tissue elasticity compresses its wall and a firm clot plugs the site of injury. Hemorrhage from these vessels occur when they are embedded in fibrotic or inflammatory tissue or when their wall is sclerotic and left without ligation. The ligation of larger vessels is a purely surgical problem. We do not discuss this point here, but we stress the obvious fact that in every series of observations one may find several cases of operative hemorrhage with perfect blood coagulation because of insufficient ligation of vessels.

This simplified but practical summary explains the causes of bleeding during surgical intervention. They are:

1. Inadequate ligation of larger vessels.

2. Capillary bleeding caused by,

a. increased capillary fragility or

b. platelet deficiency.

3. Defects in blood clotting mechanism.

What are the laboratory tests that will detect these causes preoperatively?

Cause No. 1: It is obvious that no test will forecast such accident.

Cause No. 2: a. Capillary fragility can be measured by simple tourniquet test or Petechiometer.

b. Platelet count or qualitative clot retraction.

Cause No. 3: In the presence of normal capillary fragility and platelet count, a test measuring the overall coagulability

of the blood should forecast abnormal operative hemorrhage.

Our experience with the Standard Clotting Time showed its clinical reliability not only in the present study, but also in approximately 2,500 other patients. This test is used routinely at the Kingston General Hospital for the control of anticoagulant therapy instead of prothrombin time with excellent clinical results.[4,5,6] In 29 cases of acquired or congenital disorders of blood coagulation in adults, proven by specific tests, Standard Clotting Time was significantly beyond the normal range, thus showing the abnormality in 20 or 30 minutes in contrast with the time-consuming specific tests.

In the present study, the Standard Clotting Time clearly showed the abnormal blood coagulation in every case (see Group C in Table I) in which special tests indicated a defect; furthermore, we found a considerable group of children (see Group B in Table I) with moderately prolonged Standard Clotting Time in which special tests were normal, yet in 16.2 per cent postoperative hemorrhage occurred. These clinical observations strongly suggest that the Standard Clotting Time is a more sensitive indicator of abnormal blood coagulation than any of the suggested "Special Tests." We must emphasize the important fact that our test indicates the disorder in a matter of minutes in contrast with the time-consuming special tests. It is not claimed that Standard Clotting Time shows the exact nature of the defect, and special tests may be useful to determine the site of the deficiency in the blood clotting mechanism.

Other factors in hemostasis, not measured by Standard Clotting Time as capillary fragility and platelets, can be determined easily by simple tests.

We observed only three children (see Group D in Table I) with increased capillary fragility. One bled after tonsillectomy, demonstrating the importance of this test in the routine procedure. According to our experience the properly performed tourniquet test and Petechiometer give almost identical results; however, we prefer the Petechiometer in children, because the procedure is completely painless.

Platelet deficiency can be determined by platelet count or qualitative clot retraction test. Both should be available in every hospital. In the context of our procedure, poor clot retraction (in the presence of normal Standard Clotting Time) signifies marked reduction of platelet count. All the cases in Groups A, B, and D (see Table I) had normal clot retraction or platelet count. Thrombocytopenia, however, may develop rapidly in children after apparently innocuous infections; consequently, this test should never be omitted.

A careful family history regarding bleeding conditions is important before any surgery, but our observations show that positive or negative history should not be overemphasized. A child with the history of bleeding in the family (see Table II) may have normal tests and uneventful surgery whereas a negative history does not exclude a serious defect in the blood clotting mechanism (see Table III). Our simple but meticulous tests showed that 22 out of 29 children with some history of bleeding (see Table II) had perfect hemostatic mechanism and needed no further expensive investigations. The parents were told that such disorder was not present and long persisting worries were eliminated. On the other hand, abnormal tests without suggestive history of bleeding indicated the disorder in a matter of minutes and prevented serious accidents (see Table III).

SUMMARY.

Our study of 400 children undergoing surgery showed that:

1. A careful history regarding bleeding conditions is important but has no decisive role in the management of the patient because in all cases;

2. Simple but meticulous tests such as,

a. platelet count and/or clot retraction test,

b. capillary fragility test,

c. Standard Clotting Time

should be and can be performed in every hospital and will screen out all possible bleeders.

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THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The West Virginia Academy of Ophthalmology and Otolaryngology will hold its annual meeting at the Greenbrier Hotel, White Sulphur Springs, W. Va., on April 6-8, 1961.

The guest speakers on Ophthalmology are: Dr. Irving H. Leopold, Philadelphia, Pa., and Dr. Harvey E. Thorpe, Pittsburgh, Pa.; for Otorhinolaryngology: Dr. John J. Shea, Memphis, Tenn., and Dr. F. Johnson Putney, Philadelphia, Pa.

In addition to the scientific program, arrangements have been made with Mr. Philip Salvatori of Obrig Laboratories to discuss and show techniques of contact lens fitting.

For additional information please contact the secretary, Dr. Worthy W. McKinney, 109 East Main St., Beckley, W. Va.

NON-ORGANIC HEARING LOSS.

Case Report.

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and
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On November 18, 1960, a 16-year-old boy entered the Clinic accompanied by his parents, both of whom were congenitally deaf and did not speak. This boy was referred by the Audiology Clinic at the Arkansas School for the Deaf which was temporarily out of operation. He had applied for assessment of hearing function and possible entrance to the School.

Otological examination revealed the external ear canals and drums to be essentially normal. The immediate clinical impression of this boy based on overt behavior was one of non-organic hearing loss. The patient would turn his eyes from the speaker while cupping his hand behind his ear and ask for repetitions.

During audiometric examination his responses to pure tone stimuli were very spurious as were the responses to speech reception testing. The boy repeated only one syllable of the spondaic words at a wide variety of sensation levels. Speech discrimination was slowly and laboriously done with monitored live voice, and the patient was encouraged to guess at each word. The Doerfler-Stewart test was positive.

On interviewing the boy following audiometric examination he became very anxious and distraught. He broke into tears saying that he did not wish to attend the school with hearing children, that his friends were deaf, and that he wanted to be deaf. He stated that he desired to go to the State School for the Deaf, obviously because he would feel more secure.

It became apparent that this boy's basic problem was a psychic disturbance with which he was trying to cope by feigning deafness. Clinical findings and audiometric testing indicated essentially normal hearing. He was, therefore, re-

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ferred for psychiatric study and guidance after a careful explanation to his relieved parents (by manual communication).

Approximately one week following examination, the authors received a telephone call from the family's minister. He stated concern over the boy's recent peculiar actions, especially his increased use of manual communication and decreased use of speech. The parents were also worried about the fact that their son dated only deaf girls and associated only with deaf friends.

This follow-up information further verified our diagnosis of non-organic hearing loss due to emotional disturbance and further justified the referral for psychiatric treatment.

While the patient's behavior in this case could be called conscious malingering, as he admitted to its intent, it is certainly possible that with the increased "need" for deafness on the unconscious level this could turn into a true psychogenic deafness.

The interesting feature in this case, and the reason for the presentation of this report, is the unusual educational and social barriers presented to a nonhearing family when a hearing child is born. The literature is replete with case studies and much information on the educational and social problems of deaf children born to hearing families; however, the problem encountered here is certainly not atypical among the population of nonspeaking deaf people with hearing children.

1610 West Third Street.

TOPICAL ANESTHESIA IN BRONCHOSCOPY AND BRONCHOGRAPHY.

A Report on 650 Cases with Hexylcaine.*†

And

The Little Red Tray.

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and
G. SCHRADER, M.D.,
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During their practice almost all physicians will have occasion to use regional anesthesia. Whatever the agent of choice, the fact that it is toxic to an unpredictable degree is all too frequently overlooked because of its relatively rare occurrence; in fact, anesthetics, regional or general, are neurotoxic *per se*, otherwise they would not work at all.

Once the threshold of safety is past, the patient is in an extremely hazardous state. There are few drugs which are used so thoughtlessly and with less knowledge of their untoward potentials than the topical anesthetics. Too little attention has been paid to the rate of absorption and the amount of the anesthetic. Because of the possibility of self-incrimination, due to error of technique or dosage, the mishaps are seldom reported, and, therefore, their true number is not available; these cases are probably of a higher order than one would suspect. Because of this obvious fact this report is not comparative, it is statistical only.

Compounding the problem is the fact that we have no means whatsoever of predetermining whether a patient will react

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*From Sunny Acres, Cuyahoga County Tuberculosis Hospital, Cleveland 22, O.

†Cyclaine, Merck Sharp & Dohme.

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adversely the first time he has a topical anesthetic, or at any subsequent time. Each patient who receives a topical anesthetic, regardless of whether any specific substance has been previously administered, is a potential reactor. Since we have no warning of a reaction prior to administration we must be prepared for such emergency.

TECHNIQUE OF TOPICAL ANESTHESIA ADMINISTRATION.

There are numerous methods for applying the particular anesthetic to the upper and lower portions of the respiratory mucosa. Whichever method is used, two salient points are to be kept in mind: *1.* the respiratory mucosa absorbs any anesthetic very rapidly; *2.* too vigorous administration or too much medicament may overwhelm a patient who might otherwise tolerate the product or the amount quite well. In this particular hospital, we use the spray method in administering the anesthetic. We have found this to be as efficient and adequate as any other method. Due to age, recalcitrance or anatomic resistance we have employed general anesthesia in a rare case.

For the topical anesthetic we have employed Hexylcaine in 650 cases. We use a therapeutic oral dose of barbiturate several hours prior to the operation and, at present, 25 mgm. of Chlorpromazine with 1/200 grain of atropine intramuscularly about one hour prior to the procedure.

As the administration of the anesthetic progresses, the administrator must be constantly vigilant. Should the patient begin to show signs of blanching, undue sweating, athetoid movements of the extremities, drowsiness, weakness or untoward perturbance, it is essential that the operator determine at once the patient's reaction. There are two paths, at this point, which the patient may pursue.

REACTIONS.

Central Nervous System Reaction: This is by far the most serious and may well lead to catastrophe. This type of re-

action is accompanied by violent tonic and clonic convulsions and loss of consciousness. Trying to determine the status of the pulse and blood pressure at this time is of no avail because of the convulsive state, and even a cursory neurological examination is impossible.

Unless antidotal therapy follows at once the patient may subsequently slip into a state of shock and must be treated accordingly.

Anonymous Reaction: This is a rather nebulous and non-systematized classification and includes all types of reactions which do not fall into the one mentioned above. It may be myocardial depression, circulatory collapse or both. It may be due to simple syncope, hypotension, hyperventilation or a profound response to medication given prior to the administration of the anesthetic agent. The administration may also trigger a latent allergy or epilepsy. The anonymous type of reaction seldom requires counteraction, but such procedures must be available for use if the operator's judgment so dictates. The importance of the anonymous reactions is that they be separated from the central nervous type.

Of the 650 cases receiving Hexylcaine topical anesthesia for bronchoscopy or bronchograms, we were unable to complete the examination in five instances because of reactions and for the following reasons: only one case was of the central nervous system type. The patient survived. Four cases were of the anonymous variety. These likewise recovered promptly and without antidotal therapy. The latter reactions could not be classified and, therefore, were placed in the second category. Whether any or all of these could be attributed to the anesthetic is open to question. Since they occurred following the administration of the anesthetic agent they are suspects, but only because of their concurrence.

TREATMENT OF REACTIONS.

Anyone responsible for the administration of topical anesthesia must be prepared to cope with the inevitable reaction. Not only is the physician obligated to the patient, but he also owes protection to himself from the medicolegal aspect. The

physician who does not know how to treat a severe reaction or does not have the facility immediately available for this procedure has placed himself and the patient in a potentially untenable position.

Once the operator has determined into which classification the particular reaction belongs he must realize that the two types of reaction require diametrically opposite treatment. Should one be inadvertently used against the wrong type the patient's status will become more severe.

Central Nervous System Reaction: Since this is the result of comparative overstimulation of the higher centers we must render sedation *immediately.* Inasmuch as this type of unfortunate circumstance renders the pulmonary physiology incapable of oxygenating the higher centers, any delay in giving the proper medication might lead to permanent damage in these areas.

For this purpose Secobarbital may be given intravenously into the most available vein. It is not given intramuscularly as the absorption rate might not be adequate. Other intravenous barbiturates are available and may serve the purpose just as well. The Secobarbital is given slowly until the convulsions have abated. Artificial respiration and oxygen are valuable adjuncts but are of no avail until the convulsive state has subsided. Insertion of an airway is optional.

Anonymous Reactions: These seldom require anything but anxious expectancy. Should the operator feel that something should be done for the patient he should have recourse to the following: into a 5 cc. syringe 2 cc. of Phenylephrine is withdrawn, and *then expelled from the syringe into its original container.* The needle is then placed into a vein and 5 cc. of blood is withdrawn, and then replaced. Enough of the medication has remained in the syringe and needle to produce sufficient and adequate stimulation. Insertion of an airway and administration of oxygen are optional.

COMMENT.

Five reactions were encountered in the administration of

topical anesthesia with Hexylcaine in 650 cases. Only one of these was of the convulsive type, the remaining four being classified as anonymous. It would appear from these figures that Hexylcaine has a high degree of safety.

Almost all the cases in this series were concerned with tuberculous patients. Because of increased secretions and edema of the mucosa the anesthetic agent must overcome these obstacles to be of benefit; Hexylcaine was found to be efficient and adequate for this function. We feel that one central nervous system type of reaction in 650 consecutive cases in which Hexylcaine was used shows that this product has a high co-efficient of safety.

Whatever the product used, there is a calculated risk in topical anesthesia.

The Little Red Tray is included and emphasized because of this fact.

THE LITTLE RED TRAY.

An appropriately sized red compartmented tray should be part of the mandatory equipment in all areas where regional anesthesia is administered. It may be purchased almost anywhere and is of the type used for kitchen utensils. Its contrasting color will impress the personnel because it probably will be the only red container in the area. It should contain the following: tourniquet, airway, alcohol sponges, five cc. syringes, needles, and Secobarbital and Phenylephrine in stoppered bottles.

U. S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE, PUBLIC HEALTH SERVICE, NATIONAL INSTITUTES OF HEALTH.

Travel Funds for Junior Investigators.

Funds to cover the cost of round trip air coach travel are available to junior investigators engaged in or receiving training in the neurological, otolaryngological, and related sciences who plan to attend the following international meetings:

1. Seventh International Congress of Neurology, September 10-16, 1961, Rome, Italy.

2. Fifth International Congress of Electroencephalography and Clinical Neurophysiology, September 7-13, 1961, Rome, Italy.

3. Fourth International Congress of Neuropathology, September 4-7, 1961, Munich, Germany.

4. Seventh International Congress of Otorhinolaryngology, July 23-29, 1961, Paris, France.

Requests for these travel funds can be made by letter addressed to the Executive Secretary of the Selection Committee: Dr. Adolph L. Sahs, Department of Neurology, State University of Iowa, Iowa City, Ia.

The letter should include a brief summary of the applicant's educational, occupational, and research background, and should indicate his major field of interest. The letter should be signed by the applicant and countersigned by his administrative executive.

The deadline for the receipt of requests is March 1, 1961. The Selection Committee will inform applicants of its action by April 15, 1961.

Funds for this purpose have been made available through grants to the Selection Committee from The National Institute of Neurological Diseases and Blindness, Public Health Service; and the National Science Foundation.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

PROGRAM 1960-1961.

April 4, 1961.

Parkland Memorial Hospital, Rooms No. 101-102.

5:30 P.M.—Clinical Otolaryngology and Ophthalmology Conference.

7:30 P.M.—Otolaryngology. Panel: "Hearing in Relation to Speech Formation and School Performance"—Dr. Edward Pratt, Moderator; Dr. Phillip Hood, Dr. Jack Martin, and Dr. Martha Helen Hale.

7:30 P.M.—Ophthalmology. "Pre-orbital and Adnexal Ocular Lesions with Basic Principals of Plastic Reconstructive Surgery." Panel: Dr. Billie L. Arnoff, Surgeon; Dr. Donald A. Corgill, Otolaryngologist; Dr. William C. Sellman, Plastic Surgeon; Dr. Coleman Jacobson, Dermatologist.

May 2, 1961.

Parkland Memorial Hospital, Rooms No. 101-102.

5:30 P.M.—Clinical Otolaryngology and Ophthalmology Conference.

7:30 P.M.—Otolaryngology: Dr. William Wright, Houston, Tex. Subject: "Improved Methods of Handling Bony Deviations of the Nasal Septum." Discussion: Dr. Marvin G. Shepard, Dallas, Tex.

7:30 P.M.—Ophthalmology: "Non-Surgical Considerations in Horizontal Ocular Deviations." Dr. John Lippas, Parkland Memorial Hospital.

June 6, 1961.

Parkland Memorial Hospital, Rooms No. 101-102.

5:30 P.M.—Clinical Otolaryngology and Ophthalmology Conference.

7:30 P.M.—Otolaryngology: Papers presented by Residents of our Teaching Hospitals. Dr. Ben Stegal—"Pseudomonas Infections in Otolaryngology." Dr. Sam Huggins—"Experience in Rhinoplasty Procedures." Dr. Chris Helmus—"Aortic Hemograft Reconstruction in Radical Hypopharyngeal Surgery."

7:30 P.M.—Ophthalmology: "Ocular Problems Related to Space Medicine." Speaker to be announced.

ANNUAL SPRING MEETING OF THE NEW YORK EYE AND EAR INFIRMARY ALUMNI.

The Annual Spring Meeting of the Alumni Association of the New York Eye and Ear Infirmary will take place April 17-19, 1961.

Symposia will be offered on Otosclerosis and Benign Lesions of the Larynx. There will also be a Closed Circuit Television demonstration of surgical procedures. In addition there will be lectures on Plastic Surgery, Paranasal Sinuses and Carcinoma of the Head, Neck and Larynx.

For further information write Dr. John R. Finlay, Secretary, Alumni Association, 218 Second Avenue, New York 3, N. Y.

CASSELBERRY AWARD OF THE AMERICAN LARYNGOLOGICAL ASSOCIATION.

A sufficient fund having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available in part or as a whole, for a prize award. Theses must be in the hands of Dr. Lyman G. Richards, 12 Clovelly Road, Wellesley Hills 81, Mass., Secretary of the American Laryngological Association, prior to Dec. 1 of any given year.

The Award is a prize of money with accompanying certificate signed by the officers of the American Laryngological Association. The sum of money will be agreed upon by the Council of the Association after the manuscript has been evaluated by the Award Committee. It may be awarded in whole or in part among several contestants.

Eligible contestants may be: 1. Hospital interns, residents, or graduate students in Rhinology and Laryngology; 2. An individual with an M.D. degree who is actively practicing or teaching Rhinology and Laryngology in the Americas; 3. Any scientific worker in the field of Rhinology and Laryngology.

Manuscripts shall be presented to the Secretary of the Association under nom de plume which shall in *no way* indicate the author's identity. There shall also be a sealed envelope bearing the nom de plume and containing a card showing the name and address of the contestant which the Secretary shall keep in his possession.

Manuscripts must be limited to 5000 words and must be typewritten in double spacing on one side of the sheet. The thesis shall not have been published elsewhere before submission.

The successful thesis shall become the property of the American Laryngological Association, but this provision shall in no way interfere with publication of the thesis in the Journal of the author's choice. Unsuccessful contributions will be returned promptly to their authors.

The Award which will be made at the Annual Meeting of the American Laryngological Association shall be based on:

1. Originality of material.
2. Scientific and clinical value.
3. Suitability for this Award.
4. Method of presentation as to style, illustrations and reference.

The maximum amount of the Award shall not exceed $200.00.

COURSE IN LARYNGOLOGY AND BRONCHOESOPHAGOLOGY.

The Department of Otolaryngology, University of Illinois College of Medicine, will conduct a postgraduate course in Laryngology and Bronchoesophagology from March 13 through March 25, 1961, under the direction of Paul H. Holinger, M.D.

Registration will be limited to 15 physicians who will receive instruction by means of animal demonstrations and practice in bronchoscopy and esophagoscopy, diagnostic and surgical clinics, as well as didactic lectures.

Interested registrants will please write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk St., Chicago 12, Ill.

SEVENTH INTERNATIONAL CONGRESS OF NEUROLOGY.

(Rome, 10-15 September 1961)

FIFTH INTERNATIONAL CONGRESS OF ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY.

(Rome, 7-13 September 1961).

The Seventh International Congress of Neurology will be held in Rome, September 10-15, 1961, under the auspices of the World Neurologic Federation and of the National Institute for Nervous Diseases and Blindness of Bethesda.

Together with the general Assembly of the International League against Epilepsy and the V International Congress of Electroencephalography the first day will be dedicated to the opening Session.

Monday 11 and Tuesday 12 September will be given respectively to the discussion of the I and the II Topic, while the III Topic and the communications, will be represented respectively on Thursday 14 and Friday 15. Symposia will be held on Wednesday 13 September, a day free of other Congress engagements, to permit an interval between the two parts of the works.

Registration fees and other modalities are the same as those established for the Congress of Neurology. The official banquet and dance will be held in conjunction with this Congress.

Any information is obtainable from the President of the Congress, Prof. Mario Gozzano, or from the Secretary General, Dr. Raffaello Vizioli, Viale Universita 30, Roma.

PROGRAM.

THE WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

The Fourteenth Annual Meeting of the West Virginia Academy of Ophthalmology and Otolaryngology will be held at the Greenbrier Hotel on April 6-8, 1961. The program includes:

1. Harvey E. Thorpe, M.D., Pittsburgh, Pa.: 1. Recent Developments and Personal Experiences in Cataract Surgery.

2. John J. Shea, M.D., Memphis, Tenn.: 1. Fenestration of the Oval Window After Five Years; 2. Vein Graft Tympanoplasty.

3. F. Johnson Putney, M.D., Philadelphia, Pa.: 1. Laryngeal Keratosis: A Clinico-Pathologic Problem; 2. Neck Dissection in Cancer of the Larynx.

4. Irving H. Leopold, M.D., Philadelphia, Pa., will give two lectures in which the titles will be announced later.

In addition, arrangements have been made with Mr. Philip Salvatori of Obrig Laboratories to present a session on the fitting of contact lenses.

A special convention rate will be available at the Greenbrier. There is a non-member registration fee of $25 to cover all the social and scientific sessions, which entitles graduate doctors of medicine to associate membership in the academy.

For further information write to Worthy W. McKinney, M.D., secretary-treasurer, 109 East Main Street, Beckley, W. Va., or write directly to Reservation Manager, The Greenbrier Hotel, White Sulphur Springs, W. Va.

UNIVERSITY OF TORONTO—POSTGRADUATE COURSE IN OTOLARYNGOLOGY.

On May 11, 12 and 13, 1961, there will be presented a graduate course in this specialty by the staff of the Department of Otolaryngology, assisted by two distinguished guests: Dr. Philip E. Meltzer, Interim Chief of Otolaryngology, Massachusetts Eye and Ear Infirmary, and Acting Head of the Department of Otolaryngology, Harvard Medical School; and Dr. W. G. Hemenway, Department of Otolaryngology, University of Chicago.

The first session will begin in the afternoon of May 11th, in the Royal York Hotel, Toronto, in conjunction with the Section of Otolaryngology of the Ontario Medical Association. The remainder of the sessions will be held in the clinical areas of the University of Toronto.

An attempt will be made to assess, discuss and demonstrate the newer procedures employed in the surgery of deafness. The present surgical treatment of head and neck problems will be presented with special consideration of the new conceptions of the responsibilities of our specialty in their management.

The fee for the course will be $40.00 and will include a complimentary dinner.

Please address all inquiries to the Director, Division of Postgraduate Medical Education, University of Toronto.

DIRECTORY OF OTOLARYNGOLOGIC SOCIETIES.

(Secretaries of the various societies are requested to keep this information up to date).

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Erling W. Hansen, 90 So. Ninth St., Minneapolis, Minn.
Executive Secretary: Dr. William L. Benedict, Mayo Clinic, Rochester, Minn.
Meeting: Palmer House, Chicago, Ill., October, 1961.

AMERICAN ASSOCIATION FOR CLEFT PALATE REHABILITATION.

President: Dr. J. J. Longacre, 1503 Carew Tower, Cincinnati, O.
President-Elect: Dr. D. C. Samuel Pruzansky, D.D.S., 840 So. Wood St., Chicago, Ill.
Secretary-Treasurer: Dr. Spriestersbach, Ph.D., Department of Otolaryngology, University Hospital, Iowa City, Ia.
Meeting:

AMERICAN BOARD OF OTOLARYNGOLOGY.

President: Dr. Gordon D. Hoople, 1100 E. Genesee Dr., Syracuse 10, N. Y.
Secretary: Dr. Dean M. Lierle, University Hospital, Iowa City, Ia.
Meeting: Palmer House, Chicago, Ill., October 2-5, 1961.

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION.

President: Dr. F. Johnson Putney, 1712 Locust St., Philadelphia 3, Pa.
Vice-President: Dr. Julius W. McCall, 619 Rose Bldg., 2060 E. 9th St., Cleveland 15, O.
Secretary: Dr. Daniel C. Baker, Jr., 903 Park Ave., New York 21, N. Y.
Treasurer: Dr. Charles M. Norris, 3401 No. Broad St., Philadelphia 40, Pa.
Meeting: Lake Placid Club, Lake Placid, N. Y., May 23-24, 1961 (afternoons only).

AMERICAN LARYNGOLOGICAL ASSOCIATION.

President: Dr. Edwin N. Broyles, Baltimore, Md.
Secretary: Dr. Lyman G. Richards, Wellesley Hills, Mass.
Treasurer: Dr. Francis E. LeJeune, New Orleans, La.
Editor, Historian, and Librarian: Dr. Francis W. Davison, Danville, Pa.
Meeting: Lake Placid Club, Essex Co., N. Y., May 21-22, 1961.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

President: Dr. Fletcher D. Woodward, 400 Locust Ave., Charlottesville, Va.
President-Elect: Dr. John R. Lindsay, Chicago, Ill.
Secretary: Dr. C. Stewart Nash, 700 Medical Arts Bldg., Rochester 7, N. Y.
Treasurer: Dr. K. M. Day, 121 University Pl., Pittsburgh, Pa.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 23-24-25, 1961.

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY.

Chairman: Dr. Lawrence R. Boies, Minneapolis, Minn.
Vice-Chairman: Dr. Howard P. House, Los Angeles, Calif.
Secretary: Dr. Walter E. Heck, San Francisco, Calif.
Delegate: Dr. Gordon F. Harkness, Davenport, Ia.
Alternate Delegate: Dr. Paul H. Holinger, Chicago, Ill.
Representative to Scientific Exhibit: Dr. Walter H. Maloney, Cleveland, O.
Meeting: New York City, June 26-30, 1961.

AMERICAN OTOLOGICAL SOCIETY, INC.

President: Dr. Henry L. Williams, Rochester, Minn.
Vice-President: Dr. Lawrence R. Boies.
Secretary-Treasurer: Dr. James A. Moore, New York City, N. Y.
Annual Meeting: Lake Placid Club, Essex Co., N. Y., May 26-27, 1961.

AMERICAN OTORHINOLOGIC SOCIETY FOR THE ADVANCEMENT OF PLASTIC AND RECONSTRUCTIVE SURGERY.

President: Dr. Joseph Gilbert, 111 E. 61st St., New York, N. Y.
Vice-President: Dr. Kenneth Hinderer, 402 Medical Arts Bldg., Pittsburgh, Pa.
Secretary: Dr. Louis Joel Feit, 66 Park Ave., New York 16, N. Y.
Treasurer: Dr. Arnold L. Caron, 36 Pleasant St., Worcester, Mass.

AMERICAN RHINOLOGIC SOCIETY.

President: Dr. Ivan W. Philpott, 1801 High St., Denver, Colo.
Secretary: Dr. Robert M. Hansen, 2210 Lloyd Center, Portland 12, Ore.
Annual Clinical Session: Chicago, Ill., October, 1961 (definite time and place to be announced later).
Annual Meeting: Chicago, Ill., October, 1961 (definite time and place to be announced later).

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY.

President: Dr. John J. Conley, New York, N. Y.
Vice-President: Dr. Joseph H. Ogura, St. Louis, Mo.
Treasurer: Dr. F. Johnson Putney, Philadelphia, Pa.
Secretary: Dr. George A. Sisson, Syracuse, N. Y.
Annual Meeting: Palmer House, Chicago, Ill.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY.

President: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis, Tenn.
Vice-President: Dr. John T. Dickinson, Mercy Hospital, Pittsburgh 19, Pa.
Secretary: Dr. Samuel M. Bloom, 123 East 83rd St., New York 28, N. Y.
Treasurer: Dr. Joseph C. Miceli, 879 Glenmore Ave., Brooklyn, N. Y.
Winter Meeting: February 9-10, 1961, Peabody Hotel, Memphis, Tenn.
Summer Meeting: June 28, 1961, Hotel Elysee, New York, N. Y.

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY.

President: Dr. Leland H. Prewitt, Ottumwa, Ia.
Secretary-Treasurer: Dr. Daniel S. DeStio, 121 S. Highland Ave., Pittsburgh 6, Pa.
Annual Meeting: Palmer House, Chicago, Ill., October 7, 1961.

ASSOCIACAO MEDICA DO INSTITUTO PENIDO BURNIER—CAMPINAS.

President: Dr. Alberto Gallo.
First Secretary: Dr. Alfredo Martinelli.
Second Secretary: Dr. Guedes de Melo Neto.
Librarian-Treasurer: Dr. L. de Souza Queiroz.
Editors for the Archives of the Society: Dr. Antonio de Almeida, Dr. Gabriel Pôrto, and Dr. Roberto Franco do Amaral.

ASOCIACION DE OTORRINOLARINGOLOGIA Y BRONCOESOFAGOLOGIA DE GUATEMALA.

Presidente: Dr. Julio Quevedo, 15 Calle Oriente No. 5.
First Vice-Presidente: Dr. Héctor Cruz, 3a Avenida Sur No. 72.
Second Vice-Presidente: Dr. José Luis Escamilla, 5a Calle Poniente No. 48.
Secretario-Tesorero: Dr. Horace Polanco, 13 Calle Poniente No. 9-D.

ASOCIACION DE OTO-RINO-LARINGOLOGIA DE BARCELONA, SPAIN.

Presidente: Dr. J. Abello.
Vice-Presidente: Dr. Luis Suñe Medan.
Secretario: Dr. Jorge Perellò, 319 Provenza, Barcelona.
Vice-Secretario: Dr. A. Pinart.
Vocal: Dr. J. M. Ferrando.

BALTIMORE NOSE AND THROAT SOCIETY.

Chairman: Dr. Walter E. Loch, 1039 No. Calvert St., Baltimore, Md.
Secretary-Treasurer: Dr. Theodore A. Schwartz.

BUENOS AIRES CLUB OTOLARINGOLOGICO.

Presidente: Dr. K. Segre.
Vice-Presidente: Dr. A. P. Belou.
Secretario: Dr. S. A. Aranz.
Pro-Secretario: Dr. J. M. Tato.
Tesorero: Dr. F. Games.
Pro-Tesorero: Dr. J. A. Bello.

CANADIAN OTOLARYNGOLOGICAL SOCIETY SOCIETE CANADIENNE D'OTOLARYNGOLOGIE.

President: Dr. Fernand Montreuil, 1123 St. Joseph Blvd. East, Montreal, Quebec.
Secretary: Dr. Donald M. MacRae, 324 Spring Garden Road, Halifax, Nova Scotia.
Meeting: Queen Elizabeth Hotel, June 15-17, 1961.

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. E. Hartenbower, 203 N. Main St., Bloomington, Ill.
President-Elect: Dr. Edgar T. Blair, Springfield, Ill.
Vice-President: Dr. G. LeRoy Porter, Urbana, Ill.
Delegate at Large: Dr. S. G. Baldwin, Danville, Ill.
Secretary-Treasurer: Dr. C. L. Pannabecker, Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

President: Dr. George Woodruff, Woodruff Clinic, Joliet, Ill.
Vice-President: Dr. Linden Wallner, 122 So. Michigan, Chicago, Ill.
Secretary-Treasurer: Dr. Robert Lewy, 25 East Washington St., Chicago 2, Ill.
Meeting: First Monday of each month, October through May.

CHILEAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Enrique Grünwald S.
Vice-President: Dr. Agustin Estartus.
Secretary: Dr. Marcos Chaimovich S.
Treasurer: Dr. Benjamin Kapkan K.
Director: Dr. Alberto Basterrica A.

COLORADO OTOLARYNGOLOGY SOCIETY.

President: Dr. James T. Blair, Denver, Colo.
Vice-President: Dr. James Rigg, Grand Junction, Colo.
Secretary: Dr. Will P. Pirkey, Denver, Colo.

COLUMBUS, OHIO, OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. John E. Arthur.
Secretary: Dr. M. L. Battles.
Meetings: First Monday of October through May, University Club, Columbus, O.

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Edward A. Newell.
Vice-President: Dr. Thomas M. McCrory.
Secretary-Treasurer: Dr. James L. Baldwin, 1627 Medical Arts Bldg., Dallas, Tex.

FEDERACION ARGENTINA, DE SOCIEDADES DE OTORRINOLARINGOLOGIA.

Secretary of the Interior: Prof. Dr. Atilio Viale del Carril.
Secretary of the Exterior: Dr. Aldo G. Remorino.
Secretary Treasury: Prof. Dr. Antonio Carrascosa.
Pro-Secretary of the Interior: Prof. Dr. Carlos P. Mercandino.
Pro-Secretary of the Exterior: Prof. Dr. James A. del Sel.
Pro-Secretary of the Treasury: Dr. Jorge Zubizarreta.

FIRST CENTRAL AMERICAN CONGRESS OF OTORHINOLARYNGOLOGY.

President: Dr. Victor M. Noubleau, San Salvador.
Secretary-Treasurer: Dr. Hector R. Silva, Calle Arce No. 84, San Salvador, El Salvador, Central America.

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. G. Dekle Taylor, Jacksonville, Fla.
President-Elect: Dr. Kenneth S. Whitmer, Miami, Fla.
First Vice-President: Dr. William H. Anderson, Jr., Ocala, Fla.
Second Vice-President: Dr. Marion W. Hester, Lakeland, Fla.
Secretary-Treasurer: Dr. Joseph W. Taylor, Jr., 1 Davis Blvd., Tampa 6, Fla.

FOURTH LATIN-AMERICAN CONGRESS OF OTORINOLARINGOLOGIA.

President: Dr. Dario.
Secretary:
Meeting:

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Van D. Rathgeber.
Vice-President: Dr. William Skokan.
Secretary-Treasurer: Dr. Paul Rockwell.

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. P. W. Rhyne, Albany, Ga.
Vice-President: Dr. C. L. Pennington, Macon, Ga.
Secretary: Dr. James T. King, 516 Baptist Professional Bldg., Atlanta 12, Ga.
Meeting: Oglethorpe Hotel, Wilmington Island, Savannah, Ga., March 2-4, 1961.

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. Mariano C. Caballero.
Vice-President: Dr. Joseph Freeman.
Secretary-Treasurer: Dr. H. Carlton Howard.
Meeting: Quarterly in March, May, October and December on the second Thursday of the month, 6:30 P.M., at the McAllister Hotel, Miami, Fla.

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY.

President: Dr. Jo Ono, Tokyo, Japan.
Secretary: Dr. Chevalier L. Jackson, 3401 N. Broad St., Philadelphia 40, Pa., U. S. A.
Meeting:

KANSAS CITY SOCIETY OF OTOLARYNGOLOGY AND OPHTHALMOLOGY.

President: Dr. Clarence H. Steele.
President-Elect: Dr. Dick H. Underwood.
Secretary: Dr. James T. Robison, 4620 J. C. Nichols Parkway, Kansas City, Mo.
Meeting: Third Thursday of November, January, February and April.

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. Max E. Pohlman.
Secretary-Treasurer: Dr. Wendell C. Irvine.
Chairman of Ophthalmology Section: Dr. Carroll A. McCoy.
Secretary of Ophthalmology Section: Dr. Philip D. Shanedling.
Chairman of Otolaryngology Section: Dr. Robert W. Godwin.
Secretary of Otolaryngology Section: Dr. Francis O'N. Morris.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles, Calif.
Time: 6:30 P.M. last Monday of each month from September to June, inclusive—Otolaryngology Section. 6:30, first Thursday of each month from September to June, inclusive—Ophthalmology Section.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY.

President: Dr. Arthur V. Hays.
Secretary: Dr. Edley H. Jones, 1301 Washington St., Vicksburg, Miss.
Meeting: Edgewater Gulf Hotel, Edgewater Park, Miss., May 12-13, 1961.

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

Chairman: Members serve as chairman in alphabetical order monthly.
Secretary-Treasurer: Dr. Roland H. Myers, 1720 Exchange Bldg., Memphis, Tenn.
Assistant Secretary-Treasurer: Dr. William F. Murrah, Jr., Exchange Bldg., Memphis, Tenn.
Meeting: Second Tuesday in each month at 8:00 P.M. at Memphis Eye, Nose and Throat Hospital.

MEXICAN ASSOCIATION OF PLASTIC SURGEONS.

President: Dr. Cesar LaBoide, Mexico, D. F.
Vice-President: Dr. M. Gonzales Ulloa, Mexico, D. F.
Secretary: Dr. Juan De Dios Peza, Mexico, D. F.

MEXICAN SOCIETY OF OTOLARYNGOLOGY.

President: Dr. Rafael Giorgana.
Secretary: Dr. Carlos Valenzuela, Petrarca 332-1, Mexico 5, D. F.

THE MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY.

President: Dr. Paul G. Bunker, Aberdeen, S. Dak.
First Vice-President: Dr. John A. McNeill, St. Paul, Minn.
Second Vice-President: Dr. Benjamin Bofenkamp, Minneapolis, Minn.
Secretary-Treasurer: Dr. Richard O. Leavenworth, Jr., St. Louis Park, Minn.

MISSISSIPPI VALLEY MEDICAL SOCIETY.

President: Dr. Arthur S. Bristow, Princeton, Mo.
Secretary-Treasurer: Dr. Harold Swanberg, Quincy, Ill.
Assistant Secretary-Treasurer: Dr. Jacob E. Reisch, Springfield, Ill.

NETHERLANDS SOCIETY OF OTO-RHINO-LARYNGOLOGY. (Nederlandsche Keel-Neus-Oorheelkundige Vereeniging.)

President: Dr. H. Navis, Sonsbeekweg 6, Arnhem.
Secretary: Dr. W. H. Struben, J. J. Viottastraat 1, Amsterdam.
Treasurer: Mrs. F. Velleman-Pinto, Jac. Ohrechtstr. 66, Amsterdam.

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY.

President: Dr. George T. Noel, Kannapolis, N. C.
Vice-President: Dr. J. C. Bradley, Weaverville, N. C.
Secretary: Dr. John S. Gordon, 1350 South Kings Drive, Charlotte, N. C.

NORTH OF ENGLAND OTOLARYNGOLOGICAL SOCIETY.

President: Mr. G. L. Thompson, 16 Ramshill Road, Scarborough, Yorkshire.
Vice-President: Mr. J. H. Otty, Frizley Old Hall, Frizinghall Road, Bradford, Yorkshire.
Secretary and Treasurer: Mr. R. Thomas, 27 High Petergate, York, Yorkshire.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY.

President: Dr. George C. Saunders, 644 Medical Arts Bldg., Portland 5, Ore.
Secretary-Treasurer: Dr. Donald C. Mettler, 1216 S. W. Yamhill St., Portland 5, Ore.
Meeting: Fourth Tuesday of each month from September through May, Aero Club, Portland, Ore.

OTOSCLEROSIS STUDY GROUP.

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